G. Bullock

ON

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ACUTE CEREBRO-SPINAL MENINGITIS

CAUSED BY THE

DIPLOCOCCUS INTRACELLULARIS OF WEICHSELBAUM

A CLINICAL STUDY

BY

CECIL WALL, M.A., M.D.Oxon., M.R.C.P.

(Communicated by FRANCIS WARNER, M.D., F.R.C.P.)

[*From Volume 86 of the 'Medico-Chirurgical Transactions.'*]

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ABSTRACT OF PAPER.

During the year 1901 there were admitted to the wards of the London Hospital an unusually large number of cases presenting the clinical characteristics of acute cerebro-spinal meningitis ; in many of these the nature of the infecting organism was shown by the report of Dr. William Hunter, at that time assistant bacteriologist to the hospital.

Through the courtesy of the physicians under whose care the cases were placed I have been permitted to make use of the notes, and for this permission I have to record my best thanks.

In the progress of knowledge concerning an infective disease the earliest research must necessarily be directed towards the discovery of an organism present in association with a certain definite group of symptoms ; at a later stage, if more than one organism has been isolated from different cases not easily to be distinguished in their clinical course, it becomes necessary to reverse the process and to take one organism as the constant factor and thus define the symptom group associated with its presence. The present research is the result of an endeavour to apply this method in the investigation of the pathological results of infection by the diplococcus intracellularis.

Hunter and Nuthall explained in the 'Lancet' for June 1st, 1901, their reasons for supposing that the organism they isolated from the cases in the London Hospital was identical with the meningococcus of Weichselbaum. In the present research I have endeavoured in the first place to analyse the symptoms and signs produced by infection with this organism; secondly, to discuss the pathological conditions underlying these symptoms ; and finally, to make a comparison of the results with previous descriptions of this and some other allied diseases.

It has not been thought necessary to differentiate between the cases infected by the two types mentioned by Hunter in his paper, seeing that he was satisfied as to their identity, and it was not possible to find any points

of difference in the clinical course or in the post-mortem appearances which seem to support the view that the two types were different species. If, however, other organisms were found present with the diplococcus intracellularis, the cases so infected have, for the purposes of the present investigation, been placed in a separate class.

In the absence of any trustworthy test for the presence of tuberculosis during life it has been necessary to assume that where recovery has taken place tuberculous infection was not present; this assumption seems justifiable when the clinical histories of cases undoubtedly tuberculous are compared with those of cases undoubtedly free from such infection. The Arloing-Courmont reaction has not been sought in any of the cases, chiefly because it did not seem that the test was sufficiently investigated to be considered pathognomonic.[1]

Cases which by post-mortem examination have been shown to be due to a mixed infection of the tubercle bacillus and the meningococcus have been considered separately; and many cases, from which for some reason it was impossible to obtain any cerebro-spinal fluid, have been set aside as useless in an inquiry of this nature.

In twenty-two cases presenting the clinical characteristics of meningitis, the bacteriological report renders it probable that the infection was pure and due to the diplococcus intracellularis meningitidis. Of these, twelve proved fatal, and in nine post-mortem examination proved the absence of tuberculosis; in the remaining three, of which a post-mortem examination was not possible, the clinical course seemed to exclude tuberculous infection. In nine cases, organisms other than the tubercle bacillus were present, as well as the diplococcus intracellularis; these cases are considered separately. Seven cases in which there was shown to be a mixed infection with a bacillus tuberculosis and the diplococcus intracellularis meningitidis are also reported.

[1] Horton Smith and Arnitt, 'Trans. Brit. Congress of Tuberculosis,' 1901.

TABLE I.—*Cases which died soo*

No.	Name and date.	Age.	Onset.	Duration and result.	Rigidities.	Kernig's sign.	Movements.
1	Kate A., April 4th, 1901	8 months	Fit and broncho-pneumonia	Died 3rd day	Neek rigid; legs rigid; no retraction of head	Not present	Strabismus rhythmica up-and-dow movements of eyes
2	Sarah H., Febrnary 19th, 1901	7 years	Headache; restlessness; vomited once	Died 10th day	Retraction of head; legs flaccid	Present	Intermitte strabismus twitching of face and right arm
3	Harry G., April 29th, 1901	7 years	Apathy; cough; fits with cyanosis	Died 8th day	Retraction of head	Present	Fits, gener ally right-sided
4	Joseph B., April 18th, 1901	4 years	Convulsions; screaming	Died 4th day	Retraction of head	Marked	Convulsion before admission
5	Deborah G., April 20th, 1901	2 years	Congh; no vomiting; no fits	Died 5th day	Neck rigid; head not retracted	Not present	Continuou movement of right arn and right side of face strabismus
6	Alfred C., May 26th, 1901	32 years	Right pleurisy; delirium	Died 7th day	Neck rigid; head not retracted	Present	—
7	Alfred L., April 7th, 1901	19 years	Pain in head; semi-coma	Died 12th day	Neck rigid	Present	Slight stra bismus

ter the onset of the disease.

omit.	Fundi.	Tempera-ture.	Bacteri-ology.	Meningitis.			Hydroce-phalus.	Remarks.
				Vertical.	Basal.	Spinal.		
—	—	100·5°—105°	Diplococcus meningitidis in pure culture	Patches of lymph	A little lymph	Flakes of lymph	Some excess of fluid.	
t onset only	Veins full, otherwise normal	103°—105°	Ditto	Patches of lymph; vessels congested	Purulent lymph at base	Purulent lymph all down cord	Slight excess of fluid.	
resent	? Slight neuritis	100°—103°	Ditto	—	No post-mortem.			
—	Normal	102°—104·6°	Ditto	Lymph extending up from base	Purulent lymph	Lymph on cord, chiefly in lumbar region.		
—	—	102·4°—104·6°	Ditto	Purulent lymph extending from base	Purulent lymph; excess of fluid	—	No excess of fluid	Patches of bronchopneumonia.
—	Normal	100·98°—105°	Ditto	Membranes milky	Excess of fluid	Membranes milky; no lymph	Excess of fluid	Old and recent pleurisy.
Once	Normal	100·98°—101°	Ditto	Congested lymph on frontal lobes	Lymph on superior of cerebellum	Lymph on cord.		

TABLE II.—*Cases whi*

No.	Name and date.	Age.	Onset.	Duration and result.	Rigidities.	Kernig's sign.	Movements
8	Henry R., March 27th, 1901	35 years	Severe headache; resembled enteric	Six months; death	Neck rigid; head retracted	Doubtful	Convulsion and strabismus toward the end
9	Fanny S., February 14th, 1901	2½ years	Vomiting; wasting	3½ months; death	Neck rigid; limbs rigid at times	Present	Nystagmus dissociated movements of eyes; retraction c lids
10	May P., April 13th, 1901	8 months	Sudden cyanosis; retraction of head	7 weeks; death	Head retracted	Indefinite	Slow irregular movement of limbs; nystagmus; ocular squint retraction of lids
11	Herbert C., July 19th, 1901	5 years	Diarrhœa and vomiting; headache; measles 3 weeks previously	7 weeks; death	Slight rigidity of neck	Present	R. pupil > I
12	Winifred H., July 20th, 1901	3 years	Vomiting; head-retraction followed measles	5 weeks; death	Marked rigidity of neck; legs drawn up	Absent	No strabismus; no retraction of upper lids

d later in the disease.

omit.	Fundi.	Tempera-ture.	Bacteri-ology.	Meningitis.			Hydroce-phalus.	Remarks.
				Vertical.	Basal.	Spinal.		
ttacks vom-ting sting 3—5 days	Normal	Slight pyrexia for 2 weeks at onset	Diplococ-cus menin-gitidis isolated	Convolu-tions flat-tened; hæmor-rhage into pia mater	Excess of lymph	—	Large quantity of clear fluid	
Fre-quent	Normal	Normal and sub-normal	Ditto	Convolu-tions flat-tened; lobes adhe-rent; no lymph	Excess of fluid	Some lymph or lumbar enlarge-ment		
resent owards end	Normal	Normal	Ditto	Convolu-tions flat-tened; pia arachnoid congested	—	Excess of fluid in suba-rachnoid space; pia con-gestion	Ventri-cles dis-tended; floccu-lent yellow material in ven-tricles	
Before admis-sion	Normal	101°— normal	Ditto	—	—	No post-mortem		
At first	—	104°–99°	Ditto	—	—	No post-mortem		

TABLE III.—*Cases*

No.	Name and date.	Age.	Onset.	Duration and result.	Rigidities.
13	William H., March 24th, 1901	1½ years	Irritability	10 weeks; recovery	Occasional retraction of head
14	Dorothy R., April 1st, 1901	8 weeks	Increasing size of head	12 weeks; improved	Rigidity of neck and limbs (late
15	Solomon B., April 4th, 1901	6 months	Broncho-pneumonia; convulsions	3½ months; hydrocephalus; improved	Retraction of head; opisthotonos; limbs rigid
16	Esther S., May 3rd, 1901	3 months	Constipation; convulsions; onset gradual	2½ months; recovery	Retraction of head; arms rigid
17	Ellen T., March 12th, 1901	3 months	Wasting; cough; convulsions; vomiting	5 months; recovery	Rigidity of neck
18	Esther L., April 20th, 1901	6 months	Feverish; vomiting; retraction of head	3½ months; improved	Retraction of head
19	George P., June 8th, 1901	5 years	Staggering gait	6 months; recovery	No rigidities
20	John E., July 6th, 1901	1½ years	Diarrhœa and vomiting; bronchitis	2½ months; recovery	Retraction of head
21	Albert P., August 5th, 1901	3 years	Vomiting; screaming	10 weeks	Marked retraction of head
22	John C., October 13th, 1901	3 years	Irritable	10 weeks	Retraction of head

ch recovered.

Kernig's sign.	Movements.	Vomiting.	Fundi.	Temperature.	Bacteriology.
Present	Restless movements of head and eyes	Occasional	—	98°—104°	Diplococcus intracellularis found.
resent (late)	Strabismus; retraction of upper eyelids	Late	—	Normal	Ditto.
Present	Strabismus; retraction of lids	Occasional	Normal	Normal	Ditto.
Indefinite	No retraction of upper lids; no strabismus	Occasional	Normal	Normal generally; irregular rises	Ditto.
Present	Convulsions 1 week before admission; retraction of upper lids marked	Occasional	Normal	Normal	Ditto.
Not obtained	Occasional retraction of lids; dissociated movements of eyes	Present	Normal	Normal; occasionally rose to 102°	Ditto.
Not obtained	Internal strabismus; nystagmus	Present	Early neuritis	Normal	Ditto.
Not obtained	No strabismus	—	Normal	100°—normal	Ditto.
Present	Intermittent strabismus	Present	Normal	Irregular	Ditto.
Not obtained	Dissociated movements of eyeballs	Present	—	Normal	Ditto.

TABLE IV.—*Cases of single infection, Nos.* 1—22.

Age.	Group 1. Which died early.	Group 2. Which died later.	Group 3. Which recovered.	Total.
1 year and under	2	1	2 months and under, 1 3 „ „ 2 6 „ „ 2 } 5	8
Under 2 years and over 1 year	1	0	2	3
„ 3 „ „ 2 yrs.	0	2	2	4
„ 5 „ „ 4 „	0	1	1	2
„ 7 „ „ 5 „	2	0	0	2
19 „	1	0	0	1
32 „	1	0	0	1
35 „	0	1	0	1
Total number of cases	7	5	10	22
Duration	Minimum 3 days; maximum 12 days; average 7 days	Minimum 5 weeks; maximum 6 months	Minimum 10 weeks; maximum 6 months	
Rigidity of neck	7	5	9	21
Retraction of head	3	0	8	11
Vomiting	Frequent, 1 case; once only, 2; no vomit, 3; no mention, 1	5	10	
Kernig's sign	5	3	5	13
Squint or dissociated movements of eyes	4	0	7	
Retraction of upper lids	0	2	3	
Temperature	Pyrexia, 100°—103°	Pyrexia at first, normal later, 4; pyrexia throughout, 1	Generally normal with irregular rises	
MORBID ANATOMY.				
Purulent menigitis	5 cases out of 6 post-mortems	1 case out of 3 post-mortems		
Ventricles distended with fluid	1 case out of 6	3 cases out of 3		

In all cases the bacteriological report was based upon the result of the system of examination detailed by Dr. Hunter in his paper. If there was evidence of contamination the case was excluded, so that so far as possible fallacies should be avoided.

Cases in which the Meningitis in all probability was due to a single infection by the Diplococcus intracellularis meningitidis.

Age.—Of the twenty-two cases collected, the ages varied from two months to thirty-five years. Eight were twelve months old or younger; three were between one and two years old; four between two and three; two were five years old; two seven years old; and the others nineteen, thirty-two, and thirty-five years respectively.

The condition, therefore, though occurring with greater frequency in early life, is not exclusively confined to that period. The question of age incidence is of importance in determining the relationship of this disease to posterior basic meningitis, and will be discussed later.

Source of the infection.—It has not been possible in these cases to trace the mode of infection. No instance has occurred in which there was any suspicion of transmission from person to person. In only one instance were two members of the same family similarly affected; this was in the case of Albert P— (No. 21), whose sister had been treated for posterior basic meningitis, at Great Ormond Street, in 1893.

No instance was met with in which two cases came from the same house; the homes of the parents were scattered widely through the East End of London, and in some instances the cases had been brought up from the country. There seemed nothing, in fact, to suggest that the disease was transmitted by contagion.

Where babies at the breast, hand-fed infants, children of older years, and adults seem indiscriminately affected,

it is at least improbable that the germ is introduced through the food or drink.

Bronchitic signs are not uncommon at the onset, but evidence of pulmonary disease is not invariably present. A nasal discharge is not so common as might be expected if the nose is the part through which the infection is introduced. In fact, so far as these cases are concerned, no evidence has been gathered which points conclusively to the mode of infection or to the manner of spread of the disease.

Symptoms.—When the cases are grouped together they seem naturally to fall into subdivisions, which present both clinically and pathologically definite points of difference.

Thus, to take the fatal cases, seven died in from three to twelve days from the onset of the disease, and it appears reasonable to suppose both on clinical and pathological grounds that death resulted from the severity of the meningitis. In five cases death occurred in from five weeks to six months from the commencement, and for many reasons the supposition seems justifiable that the fatal event was determined not by the actual meningitis, but by conditions consequent upon it. Closely allied with this second group of cases are those which terminated in recovery either partial or complete.

In discussing the symptoms, therefore, it is necessary to distinguish so far as is possible between those which are indicative of actual meningitis and those indicative of secondary conditions, of which the most frequent and most important are the excess of cerebro-spinal fluid and the distension of the cerebral ventricles.

Onset.—Of the seven cases which died early in the disease, three showed evidence of pulmonary disease when first seen; two seemed at first to be cases of bronchopneumonia; the third commenced with the symptoms and signs of a dry pleurisy. Of the other four, two began with severe headache, one with convulsions, one with incontinence of urine followed two days later by a succes-

sion of fits. Of the five cases which died later in the disease, in two the first sign of meningitis was headache occurring two or three weeks after an attack of measles, in one the onset was sudden, with what the mother described as "inward fits," and in the two remaining the onset was gradual, in one case resembling the onset of enteric fever, and in the other with vomiting and wasting. Of the ten cases which recovered, in five the onset was gradual, and it was not possible to fix closely the date when the illness first commenced; in three, convulsions ushered in the disease, in one irritability and anorexia were the first symptoms suggesting departure from health, and one began with diarrhœa, vomiting, and some bronchitis.

From a consideration of these points it seems clear that the development of symptoms and signs may in some cases be extremely gradual, though in the majority the ingravescence of symptoms is rapid as soon as the disease starts. In one case, Dorothy R— (No. 14), the disease had obviously progressed far, as evidenced by the enlargement of the head, before any symptoms were developed. As a general rule, however, the rapid development of symptoms and signs shows a considerable difference from the insidious onset of tuberculous meningitis, and is, therefore, of diagnostic importance.

Pulmonary disease somewhat frequently, but not invariably, precedes the symptoms of meningitis; when the frequency of bronchitis and broncho-pneumonia amongst East End children is considered, it is dangerous to lay much stress on pulmonary complications when considering the etiology of meningitis.

Rigidity of the neck.—The most marked sign, and that which most frequently first suggested the diagnosis of meningitis, was the rigidity of the neck, or rather resistance to forward flexion of the head. In one case only (George P—, Case 19) out of the twenty-two investigated was this sign absent, and he had been ill for four or five months before admission to the hospital.

In many cases there was very marked retraction of the head, producing the condition of cervical opisthotonos; but in others the rigidity, though marked, was not associated with much retraction. As a rule there was no resistance to rotation of the head, and difficulty in movement was only encountered when forward flexion was attempted.

Spinal opisthotonos was not marked in any of these cases; most frequently the patient lay upon the side with full flexion of the spine, even when the head retraction was so marked that the occiput seemed to lie between the scapulæ. In other cases that I have seen since, spinal opisthotonos was extreme.

In the cases that recovered, the rigidity of the neck, though it might vary from day to day, was usually one of the last signs to disappear.

Kernig's sign.—In the investigation of this sign of spinal meningitis, certain fallacies are likely to creep in which are very difficult to exclude. In the original description the sign is demonstrated by bringing the patient to the sitting posture and then attempting to straighten the knees. In a modified form, described by Professor Osler in the Cavendish Lecture, the patient remains in the dorsal decubitus; both hips are then flexed to a right angle with the trunk, and an attempt made to fully extend the knees. In well-marked cases the resistance of the hamstrings is so great that extension of the knees beyond a right angle is impossible. Osler's modification is advantageous in so far as it disturbs the patient but little.

In the less marked cases, however, with this method a fallacy may be introduced from tilting of the pelvis and flexion of the lumbar spine: the knees may be fully extended, but the flexion of the hips has been decreased so that no increase of tension is brought to bear upon the hamstrings.

To avoid this fallacy it is necessary to keep one hip fully extended, while the other is kept flexed to a right

angle, and the knee-joint is gradually extended. In this way the sign may be elicited without greatly disturbing the patient. Further, the presence of the sign, especially in adults, cannot be considered as pathognomonic of spinal meningitis. In cases of true sciatica, for instance, it can be very readily demonstrated, and in many perfectly healthy individuals full extension of the knee-joints, when the hips are flexed to a right angle, is a distinctly uncomfortable, if not impossible proceeding. Not many adults can perform the schoolboy trick of touching their toes without bending the knees. Sailer, too, has shown that it may occur in one leg in cases of focal brain disease.[1]

The question also arises concerning the degree of limitation in extension necessary before the sign can be said to be definitely present.

For the present purpose a rough limit of one and a half right angles has been taken. If, when the hip is flexed to a right angle and the other hip is fully extended, extension of the knee-joint can be effected beyond 135°, it has been taken that Kernig's sign was not definitely present.

The value of the sign seems to be somewhat doubtful; it does not seem possible to assert that the presence of this sign will establish the diagnosis of a case in which the other signs are equivocal. In five out of the seven cases that died in the acute stage of the disease the sign was present; in two it was definitely stated to be absent. In three out of the five which died later it was also present, and in five out of the ten that recovered it was obtained. In some cases it seemed to be variable, being only obtained occasionally.

Mental disturbance.—In those patients old enough to permit observations on this point an early and profound disturbance of the mental state was noticed. Sometimes at first there was very marked irritability and great resentment against any disturbance; this in the acute cases generally passed into a condition of drowsiness or semi-

[1] Sailer, 'Amer. Journ. Med. Science,' May, 1902.

coma, in which the patient took no notice of any interference, and, later, into a more profound coma preceding death. In those cases in which the clinical course was protracted the progressive mental deterioration could be more closely observed, and in those that recovered the inverse sequence of semi-coma, irritability, and "impaired cerebration" was often noticed.

There are probably two ways in which the functions of the brain may be disturbed in cases of meningitis. Early in the disease, associated with the meningitis there is a degree of inflammation of the cortex which gives origin to the rapidly developing coma. In this respect simple meningitis differs from the tuberculous form in which the mental faculties may be preserved until a comparatively late period of the disease. At a later stage of the disease, if hydrocephalus ensues, the brain functions may be mechanically disturbed by the increase of intra-cranial pressure. Sometimes there may even be a period of lull, or perhaps amelioration of symptoms, denoting the subsidence of inflammation, and prior to the development of hydrocephalus.

Either of these conditions, if the case be not fatal, may leave some degree of mental defect as a permanent legacy.

Movements.—At all stages of the disease it is common to find evidences of disturbance of the motor side of the nervous system. In young children convulsions may occur at the onset, in older patients they may only be an expression of some secondary complication, but in one case (Harry G—, No. 3) right side epileptiform fits were a prominent feature from the onset. From the commencement, dissociated movements of the eyeballs or an intermittent strabismus, in some cases producing a coarse kind of nystagmus, are not infrequently found. A permanent strabismus suggestive of nerve paralysis appears to be rare. In four out of the seven acute cases a squint was noticed; in one there was continuous rhythmic up-and-down movement of the eyes. Uncontrolled move-

ments of a monoplegic or hemiplegic distribution were noticed in two of the seven acute cases.

Later in the disease, and possibly associated with the supervention of internal hydrocephalus, it is common to find intermittent retraction of the upper eyelids, so that a line of sclerotic is shown above the cornea. This has been described as comparable to Stellwag's sign in Graves' disease, but it is not associated with any exophthalmos.

Epileptiform convulsions also may occur in the later stages of the chronic form, and probably are to be associated with the hydrocephalus rather than with the meningitis.

Temperature.—In the acute cases and at the onset, the temperature was generally raised, varying between 100° and 105°. Occasionally the temperature became very high just before death. In the chronic cases that were admitted shortly after the onset of the disease some irregular pyrexia was generally noticed at first, but later the temperature continued normal or subnormal. Sometimes, especially in young children, the temperature became markedly irregular in later stages when the intra-cranial pressure was high. The temperature chart differs as a rule from that of a case of tuberculous meningitis in so far as there is considerable pyrexia at the onset and during the first week or ten days, and then frequently a long apyrexial period. In tuberculous meningitis, on the other hand, the temperature is generally only slightly raised at first, and then in a more uniform manner.

Wasting.—Loss of flesh was a very marked feature in all cases, and seemed to be much more rapid than is usual in cases of tuberculous meningitis.

Vomiting.—In the acute cases vomiting was infrequent; in one only out of the seven cases did the vomiting become at all urgent. Two vomited once; the remaining four did not vomit at all.

On the other hand, in the more protracted cases,

whether they ended in death or recovery, vomiting became a marked feature as the disease progressed. The operation of lumbar puncture seemed not infrequently to have an immediately beneficial effect in checking this symptom. The necessary deduction seems to be that the vomiting is due rather to a rise of intra-cranial pressure than to the actual meningitis.

Action of the bowel.—In the majority of the cases, whether of the acute or chronic type, there was no tendency to constipation. Frequently actual looseness of the bowels was observed. In one case only out of the twenty-two (H. W. R—, No. 8) was there marked constipation, and this was the case that had a history of six months' illness before admission to the hospital. This point, though apparently unimportant, may serve at times in assisting the differentiation of this disease from tuberculous meningitis.

Pulse.—No special peculiarity has been noticed with regard to the pulse; in the acute stages the frequency was increased, but the phases met with in tuberculous meningitis were not observed. With the rise of intra-cranial pressure that appears to occur in the protracted cases, it seemed reasonable to expect a marked reduction in the pulse rate. This, however, has not been found. Irregularity of the rhythm was noticed, sometimes associated with marked severity of the other symptoms.

Respiration.—As a rule there was no marked alteration in the respiratory rhythm. Towards the end, in some of the bad cases, a grouped or periodic rhythm was noticed.

Fundi.—In twenty out of the twenty-two cases no alterations in the fundi were noticed. In one of the acute cases it was thought that there might be some early optic neuritis, but the observation was considered doubtful. In one of the cases that recovered a condition was found which was stated definitely to be early optic neuritis.

The changes in the disc described by Thursfield,

'Lancet,' February 16th, 1901, as occurring in posterior basic meningitis, though sought for, were not observed in any of the cases.

Blindness was apparently present in some of the cases without recognisable changes in the fundi; this disappeared later as convalescence was established.

Observations upon vision are extremely difficult to make when a patient is seriously ill, and consequently no extended research upon this point could be made.

Hearing.—In some of the cases, at certain periods of the disease, deafness seemed to be present, though in none was it permanent. Sometimes the attention of the patient could be attracted by sight where sounds seemed to produce no effect. Taste and smell could not be investigated.

Hydrocephalus.—In two cases in which the onset of the disease occurred before the synostosis of the cranial bones, definite enlargement of the skull was noted (Dorothy R—, No. 14, and Solomon B,— No. 15; and H. W. P—, Appendix II). In other cases the increase of fluid in the cerebral ventricles could only be surmised during life from the presence of certain symptoms which have already been discussed.

Bulging of the fontanelle, if still unclosed, was present in several of the cases, and seemed to be an indication of internal hydrocephalus. The bulging was generally relieved if a successful lumbar puncture was performed, *e. g.* Solomon B—, No. 15.

Morbid anatomy.—Of the acute cases, all save one (Alfred C—, No. 6) showed diffuse purulent lepto-meningitis of brain and cord; the greatest collection of lymph was usually at the base, but there was, in all these cases, scattered lymph upon the vertex and down the spinal cord. In the single exception there was an excess of fluid in the ventricles and a milkiness of the membranes, but no definite lymph. In this group of cases the ventricles, though containing an excess of fluid, did not appear so distended as in those cases in which the disease ran a more protracted course.

TABLE V.—*Cases*

No.	Name and date.	Age.	Onset.	Duration and result.	Rigidities.	Kernig's sign.	Movements.	Vomiting
23	Elizabeth S., March 8th, 1901	6	Fell down-stairs; headache; vomiting began 5 days later	13 days; death	Rigidity of neck	Present	Fit; irregular movements left hand; nystagmus; dissociated movements of eyes	At onset only
24	Bella C., March 11th, 1901	1 month	Convulsions	2 days; death	Rigidity of neck	No note	Continuous convulsions	No note
25	Michael G., April 4th, 1901	4 months	Fits	8 days; death	Rigidity of neck (late)	Not obtained	Repeated convulsions	Nil
26	Samuel G., April 2nd, 1901	15 years	Headache	25 days; death	Rigidity of neck	—	—	Nil
27	Isaac M., May 7th, 1901	19 years	Sudden onset; drowsiness; headache; vomiting; diarrhœa	8 days; death	Neck rigid; slight retraction of head	Present later	Delirious; restless at first, stupor later; intermittent strabismus	Occasional
28	Jessie B., May 7th, 1901	1¼ years	Sudden onset with fit	25 days; death	Retraction of head	Not obtained	Frequent yawning; no squint; no retraction	At end frequent
29	Abraham D., January 30th, 1901	2 years 10 months	Irritable attacks of cramp; fits; wasting	3 months; death	Retraction of head marked	Not obtained	Strabismus; occasional retraction of upper lids	Frequent
30	Edwin T. R., March 2nd, 1901	4½ months	Convulsions	4 months; death	Retraction of head marked	Not obtained	Occasional retraction of upper lids; no strabismus	Only during last week
31	Harry K., February 28th, 1901	1 year	Vomiting; fever; broncho-pneumonia	About 6 months; recovery	Retraction of head marked	Present	Strabismus; movements of lower jaw; later occasional retraction of upper lids	3rd and 6th weeks frequent

l infection.

di.	Temperature.	Bacteriology.	Meningitis.			Hydrocephalus.	Remarks.
			Vertical.	Basal.	Spinal.		
o nite ages	99°—101°; 102·4° just before death	Diplococcus intracellularis and pyogenic organisms	No lymph	Lymph at base and on cerebellum	Lymph down cord	Excess of fluid in ventricles at base and in spinal theca	Tubercle of the pericardium by direct extension was found, but apparently had not caused death.
-	100·4°	Ditto	Purulent meningitis.				
--	100°	Ditto	No evidence of meningitis	—	—	No excess of fluid.	
mal	99·4°	Ditto	Convolutions flattened; no lymph	Much fluid; no lymph	—	Ventricles distended	Otorrhœa 5 years, ceased just before onset of symptoms; no mastoid signs.
nal; nd; af	99°—130°	Diplococcus intracellularis and pneumococcus	Streaks of lymph	Lymph on under surface of cerebellum	Lymph all down cord	No excess of fluid; lymph in 4th ventricle	Developed signs of left basal pneumonia; post mortem, double apical and left basal pneumonia.
th es red	100°—103°, irregular, sustained	Ditto	No lymph	Purulent basal meningitis	Spinal meningitis purulent	No excess of fluid in ventricles	Signs of general broncho-pneumonia; post mortem, general broncho-pneumonia.
tic itis	Normal	Diplococcus intracellularis and *bacillus influenzæ*	No signs of meningitis; convolutions flattened	No sign of meningitis; membranes at base turbid	—	Ventricles enormously distended.	
mal	Normal	Ditto	No signs of meningitis; convolutions flattened	Milkiness of membranes; no lymph	Much fluid in theca; no lymph	Ventricles enormously distended	Improved slightly for some time; diarrhœa and vomiting for last week before death.
ar- ly for ac e; nal	Irregular for 3 weeks, 97° —104·2°, later normal with occasional rise:	Ditto.					

TABLE VI.—*Cases of mixed infection, Nos.* 23—31.

Age.	Group 1. Which died early.	Group 2. Which died later.	Group 3. Which recovered.	Total.
1 year and under . .	2 (1, 1 month; 2, 4 months)	1 (4½ months)	1	4
Under 2 years and over 1 year	1	0	0	1
„ 3 „ „ 2 yrs.	0	1	0	1
6 „ . .	1	0	0	1
15 „ . .	1	0	0	1
19 „ . .	1	0	0	1
Total number of cases .	6	2	1	9
Duration . . .	Minimum, 2 days; maximum, 25 days; average, 13½ days	1, 3 months; 2, 4 months	6 months.	
Rigidity of neck . .	6	2	1	9
Retraction of head . .	2	2	1	5
Vomiting . . .	3 slightly	2 towards end	1 (3rd—6th week)	6
Kernig's sign . .	2	0	1	3
Squint or dissociated movements of eyes	2			
Retraction of upper lids .	—	2	1	3
Temperature . .	Pyrexia	Normal	Irregular first 3 weeks; normal later.	
Purulent meningitis .	4 cases (6 post-mortems)	0 (2 post-mortems).		
Ventricles distended with fluid	2	2		

Post-mortem examination was only permitted in three out of the five cases which died at a later stage of the disease.

In these three cases the characteristic feature was the distension of the ventricles and the flattening of the convolutions. In all three a little yellow lymph was found in the descending horn of the lateral ventricles, floating in clear fluid. In one there was some lymph at the base of the brain; in another, some lymph found only on the cord most marked at the lumbar enlargement. In the third no lymph was found upon the meninges, but only some matting together of the convolutions.

Cases in which there was a Mixed Infection.

In four cases cultures from the cerebro-spinal fluid were found to contain the diplococcus intracellularis together with pyogenic organisms.

In two it was accompanied by the pneumococcus, and in three by a short bacillus resembling the bacillus influenzæ.

In those cases where pyogenic organisms or the pneumococcus were present the clinical course corresponded to the acute type of pure meningococcus infection, death occurring between the second and the twenty-fifth day.

In one case (Michael G—, No. 25) there was no evidence of meningitis at the post-mortem examination, and in another (No. 26) there was only hydrocephalus without apparent meningitis; in the other three the appearances were those of acute leptomeningitis.

In the three cases in which the Bacillus influenzæ was present the course was much more protracted; two died at the end of three and four months respectively, with post-mortem appearances suggestive of hydrocephalus as the cause of death.

The third (Harry K—, No. 31), though at one time showing signs suggestive of a marked rise in the intra-

cranial pressure, after six months made an apparently complete recovery.

The analysis of the symptoms points to the close relationship of this group with those where the infection was shown to be pure.

Rigidity of the neck is constant and appears early. In some there is definite retraction of the head ; in all cases there is impairment of the cerebral functions, though in the younger patients it is impossible to distinguish this from the peevishness very generally associated with ill-health. Kernig's sign is not constant. Occasionally dissociated movements of the eyes are found, producing an intermittent squint or even a coarse nystagmus. At the onset the temperature is usually raised, but as a rule to no great height. With the onset of internal hydrocephalus, vomiting and retraction of the upper lids are signs that become prominent.

Cases infected by Bacillus tuberculosis and Diplococcus intracellularis.

In six cases, the cerebro-spinal fluid of which during life was reported to contain the diplococcus intracellularis, acute miliary tuberculosis was found present at the autopsy.

In these the clinical course seemed to be somewhat longer than in the simple acute cases ; the average duration from the onset was twenty days, but none lived longer than twenty-six days.

Rigidity of the neck was a constant phenomenon, and dissociated movements of the eyeballs were noted in five out of six.

Pyrexia was a more noticeable feature than in those where there was no tuberculous infection, and vomiting was much more commonly present.

Save for the rigidity of the neck, the cases more nearly resembled tuberculous meningitis than those infected by the diplococcus intracellularis.

In addition to the cases here reported, there were admitted to the London Hospital during the year 1901 seventeen cases of tuberculous meningitis, as shown by post-mortem examination, the cerebro-spinal fluid of which proved sterile.

Further, there were thirty-five other cases admitted presenting the clinical characteristics of acute cerebro-spinal leptomeningitis, but of which it was not possible to determine the infecting organism; of these, sixteen recovered and were discharged, and nineteen died.

Fourteen of the fatal cases were submitted to post-mortem examination, and in twelve purulent meningitis was found present, which had apparently arisen apart from any local source of infection; in one there was acute hydrocephalus without any sign of tuberculosis, and in one the condition was described as acute hæmorrhagic leptomeningitis.

Several of these cases were doubtless due to infection by the pneumococcus or by pyogenic organisms, but many seemed in all probability to be cases akin to those infected by the diplococcus intracellularis. They are mentioned to show the frequency with which acute leptomeningitis occurred during the year.

TABLE VII.—*Cases of mixed infection.*

No.	Name and date.	Age.	Onset.	Duration and result.	Rigidities.	Kernig's sign.	Movements.	Vomiting.
32	William A., Sept. 17th, 1901	3 years	Vomiting; constipation	26 days; death	No rigidity of neck at first; rigid later	Indefinite	Dissociated movements of eyes; restless	None in hospital
33	William W., March 13th, 1901	1¾ years	Irritability; retraction of head; convulsions	26 days; death	Rigidity of neck; at times retraction of head	Present sometimes	Convulsive movements of left arm and leg	Just at end
34	Ethel G., April 15th, 1901	3½ years	Headache for months; vomiting 5 days; constipation	10 days; death	Slight rigidity of neck; some rigidity of limbs	Not present at first; definite later	Occasional strabismus; convulsive movements of left arm and leg, and less so right arm	First week
35	Charles F., April 16th, 1901	½ year	Vomiting; looked ill	14 days; death	Retraction of head; opisthotonos marked; limbs rigid later	Not present	Convulsions; dissociated movements of eyes	Frequent
36	John B., April 20th, 1901	3 years	Cough; fever; diarrhœa	23 days; death	Neck rigid; limbs rigid	Present on left side	Nil	At first
37	Lily G., April 2nd, 1901	10 months	Vomiting and diarrhœa; squint later	18 days; death	Neck rigid	Not present	Strabismus	Throughout
38	Ellen H., June 17th, 1901	5 years	"Tired;" headache; vomited once	24 days; death	Neck rigid	Present	Strabismus; dissociated movements of eyes; irregular movements of limbs	Once

...uberculosis *and* Diplococcus intracellularis.

...di.	Temperature.	Bacteriology.	Meningitis.			Hydrocephalus.	Remarks.
			Vertical.	Basal.	Spinal.		
—	Normal; rose to 100·2°	Diplococcus found in fluid from lumbar puncture	Tubercles along vessels; no lymph	Much lymph at base and under cerebellum	—	Excess of fluid; some lymph in ventricles	Caseous mediastinal gland; no other evidence of tuberculosis.
...mal	100°; rose to 108° before death	Ditto	—	Tubercular meningitis	—	—	Had been attending the hospital 1 year for congenital syphilis; post mortem, miliary tuberculosis of all organs.
...mal	99°—102·8	Ditto	—	Much lymph; tubercles along vessels	No lymph; excess of fluid (clear)	Slight excess of fluid in ventricles	Miliary tuberculosis of all organs; caseating mediastinal gland.
...mal	99°—100°; 102·4° later	Ditto	—	Lymph at base with tubercles	Tubercles on spinal membranes	Excess of fluid; convolutions flattened	Caseating bronchial glands; miliary tuberculosis of lungs and spleen.
—	Sustained 100°—104°	Ditto	No lymph	Much lymph at base; many tubercles	Excess of fluid; no tubercles; no lymph	Ventricles distended	Caseating bronchial glands; miliary tubercles of lungs and spleen.
—	97°—98°	Ditto	—	Membranes thickened; tubercles along vessels	—	No excess of fluid	Caseating bronchial gland; miliary tubercles in spleen.
...rmal	100°—101°	Ditto	Vertical meninges; œdematous	Tubercular meningitis	—	Excess of fluid in ventricles	Tuberculous focus in left lung; miliary tuberculosis, lungs, pericardium, and spleen, kidneys, liver.

TABLE VIII.—*Cases of mixed infection by B. tuberculosis and Diplococcus intracellularis.*

Age.—6 months, 1 } „ 10 „ 1 }	2
„ Over 1 year and under 2 years	1
„ „ 2 years „ 3 „	2
„ „ 3 „ „ 4 „	1
„ „ 4 „ „ 5 „	1
Total	7

Duration.—Minimum, 10 days; maximum, 26 days; average, 20 days.
Rigidity of neck.—All cases.
Retraction of head.—2 cases.
Vomiting.—6 cases.
Kernig's sign.—3 cases.
Squint or dissociated movements of eyes.—5 cases.
Retraction of upper lids.—No cases.
Temperature.—6 cases with pyrexia.

Some Points in the Pathology of Acute Cerebro-spinal Meningitis.

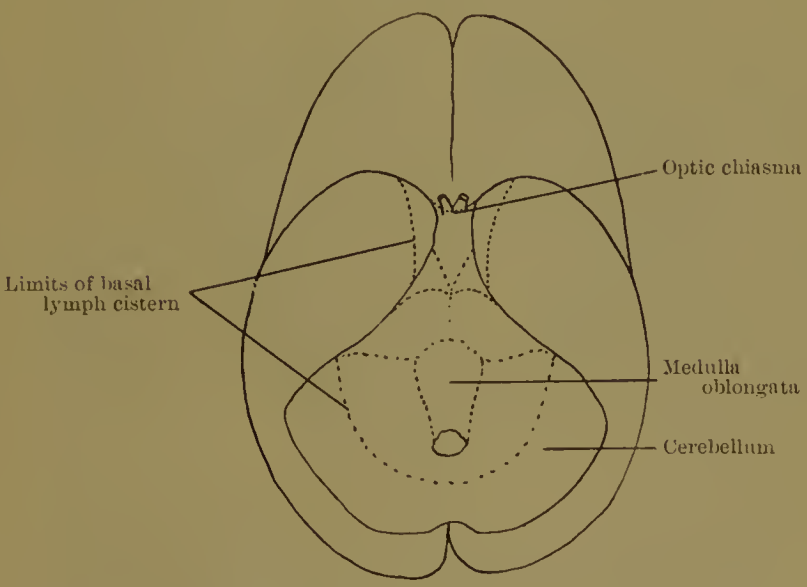
Dr. Carr, in his paper on "Posterior Basic Meningitis," has dealt at some length with the causal relationship of certain pathological conditions to the clinical characteristics.

The rigidity of the neck, early convulsions, rigidity of the limbs, twitchings and uncontrolled movements he ascribed to the cortical irritation produced by the meningitis.

In this connection the boundaries of the so-called posterior arachnoid cistern are of some importance. From the point of view of comparative anatomy it is not yet decided whether the arachnoid should be considered as a membrane distinct from the pia mater, or whether the two structures should be considered as one. The morbid anatomy of meningitis shows that pus or fluid may collect in a space bounded by two limiting membranes, known commonly as arachnoid and pia mater, which are connected together by numerous bridles. Over the greater part of the surface of the brain these bridles are short

and the space is merely potential; in other parts—for instance, the fissures of the cortex or the superior surface of the corpus callosum—the bridles are longer, and an actual space is present in which fluid may collect. If the fluid be clear the appearance suggests œdema of the cortex; if purulent it is termed purulent meningitis. At the base of the brain there are certain places where these bridles are much elongated, and thus are produced the arachnoid cisterns of Key and Retzius. In basic meningitis it is found that there is a large collection of fluid in

Fig. 2.

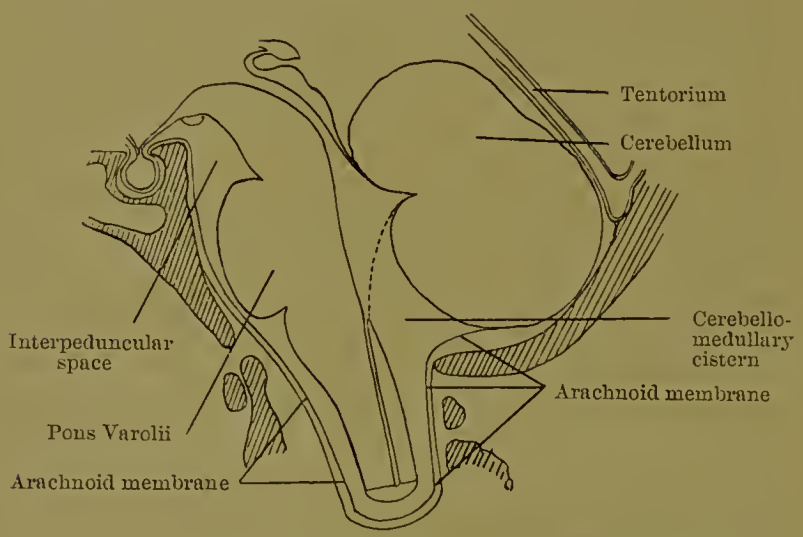


The brain viewed from below to show the limits of the great arachnoid space when distended with lymph.

these cisterns, which are in such free communication that they appear to form one large space bounded by the line of firmer attachment of the arachnoid to brain or cerebellum. Anteriorly the arachnoid seems to be firmly attached along the inner margins of the temporo-sphenoidal lobes, except just at the anterior poles, where the line of firm attachment is somewhat farther out, and from these boundaries to form a kind of bridge over the optic chiasma, the interpeduncular space, and the anterior sur-

face of the pons; below, the space thus formed is continuous with the subarachnoid space of the cord. Laterally the arachnoid is loosely attached round the cerebellar peduncles and over the lobus centralis on the superior surface of the cerebellum, a space being formed which becomes continuous with that on the superior surface of the corpus callosum. Posteriorly the great basal space thus formed passes round the medulla and becomes the cisterna cerebello-medullaris, limited behind when the arachnoid becomes again united to the pia mater on the

Fig. 3.



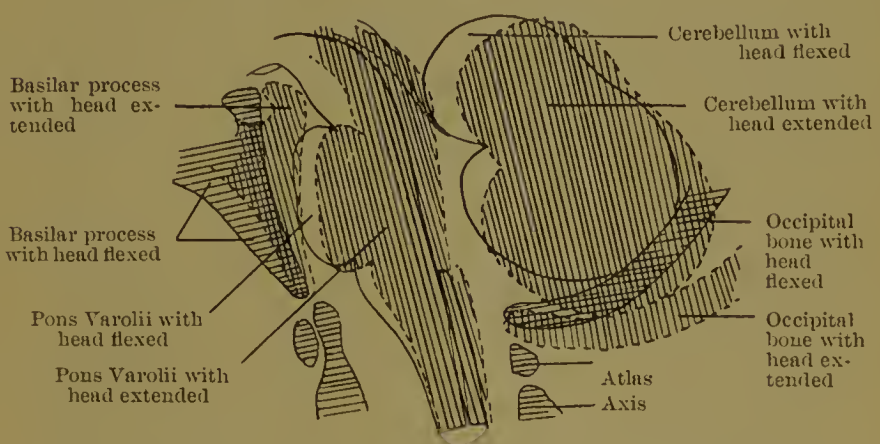
To show the communication of the basal arachnoid cisterns with the spinal subarachnoid space. The arachnoid spaces are shown as if much distended.

under surface of the cerebellum at some distance from the anterior extremity of that organ. The spaces thus marked out can be clearly recognised when filled with purulent material, and, by means of a probe, the situations in which the bridles become shorter can easily be demonstrated, although the greater part of the arachnoid bridge is necessarily destroyed in removing the brain from the skull.

The coloured plate in Allbutt's 'Medicine,' vol. viii, p. 496, shows well the limits of these basal cisterns.

The existence of the cerebello-medullary space is possibly to be explained as an arrangement to permit free antero-posterior movement of the head. In such movement there must necessarily be some sliding of the lower surface of the cerebellum over the medulla: the loose attachment of the arachnoid permits this movement. This sliding of the cerebellum up and down the medulla can easily be demonstrated by removing the bones from one side and base of the skull, and taking out one lateral lobe of the cerebellum. The medulla is thus exposed from the side, and the movement can readily be observed. It can

FIG. 4.



To show the movement of the cerebellum in relation to the medulla in antero-posterior movements of the head. The size of the spaces has been much exaggerated.

be seen almost equally well if, after removal of the cerebrum, a large window be made in one side of the tentorium, and the lateral lobe of the cerebellum be removed through this, so that the medulla is exposed to view. The rigidity of the neck or retraction of the head present, when there is posterior basic meningitis, may be looked upon as protective and preventing mechanical disturbance of the inflamed arachnoid, and comparable in explanation to Kernig's phenomenon. In this connection it is interesting to note that in such cases there is seldom any resistance against rotation of the head round a vertical axis.

The presence of the arachnoid cisterns at the base of the brain permits the collection of lymph in that situation; the subarachnoid space over the vertex is much smaller, and the facility for accumulation of inflammatory products much less. When the convolutions become flattened, in consequence of the rise of intra-ventricular pressure, the space must be still further narrowed. Thus, perhaps, should be explained the predominance of the signs of inflammation at the base when simple meningitis has passed the earliest stage.

This accumulation of inflammatory products in the basal arachnoid cisterns has suggested the use of the term "posterior basic meningitis" to signify the condition. There is no reason, however, to suppose that the inflammation is confined to that region, though the products accumulate there. Many cases which in their clinical characters agree with those described as posterior basic meningitis, show this basal collection of lymph, but also show distinct evidence of inflammation of the vertical meninges, and also of those of the spinal cord. In the circumstances, unless it can be proved that posterior basic meningitis is a distinct and independent disease, it seems best to consider such cases as examples of acute general leptomeningitis in which the inflammatory products have for the greater part accumulated in the basal arachnoid cisterns.

Carr ascribes the strabismus to implication of one or other of the oculo-motor nerves in the inflammatory process. When the extent of the basal inflammation is considered, this would seem to be extremely probable. It is curious, however, how seldom a permanent paralytic squint seems to occur; intermittent strabismus, at times described as a coarse nystagmus or merely as dissociated movements of the eyeballs, is extremely common. Two explanations of this phenomenon offer themselves. Either —owing to depression of the cerebral centres—there is a reversion to a condition existent at an earlier stage of development, an explanation usually offered to explain the

dissociated movements of the eyes of children during sleep, or of most young people during chloroform narcosis; or else it is due to the irritation of cortical or subcortical centres by the inflammatory process, and therefore comparable to the irregular movements of the limbs frequently met with in this disease.

In accordance with the view that the squint is not due to implication of the oculo-motor nerve-trunks in the inflammatory process, is the extreme rarity of affection of any other cranial nerve.

Dr. Carr has discussed at considerable length in his paper the views that have been urged with regard to the pathological conditions underlying hydrocephalus.

He takes exception to the view that the increase of the intra-cerebral fluid is due in all cases to the blocking of the foramen of Magendie, and urges that in some cases, as originally described by Merkel, an excess of fluid is found in the spinal meninges.

In the cases here recorded this has been the rule.

It was generally found that there was a slight excess of fluid in the cortical subarachnoid space, producing an œdematous appearance of the meninges, a greater collection at the base in the great arachnoid cistern, the posterior arachnoid cistern, and also in the spinal subarachnoid space.

Excess of fluid in these situations cannot be explained on any mechanical hypothesis, and it seems unnecessary to suppose that the intra-cerebral collection of fluid has a cause different from the extra-cerebral collection. Clinically the effect of withdrawing fluid by lumbar puncture in reducing the tension of a bulging fontanelle, and in the amelioration of symptoms, suggests that there is not in every case an obstruction at the foramen of Magendie.

Finally, if obstruction of the foramen of Magendie were a common cause of hydrocephalus, it would not be unnatural to suppose that in some cases at least dilatation of the central canal of the cord would occur. In none of the cases here reported was this condition found, nor does literature seem to contain records of such cases.

This mechanical view of the pathology of hydrocephalus has been urged against the employment of lumbar puncture as a remedial measure. It seems, however, that this particular argument against the mode of treatment is based upon a fallacy.

A second mechanical view, originally urged by Rilliet and Barthez and subsequently by Bastian, and discussed by Carr, suggests as a cause of the exudation an obstruction of the veins of Galen. In several cases where there was much lymph at the base in the great arachnoid cistern, it was found that this lymph extended round the peduncles of the cerebellum to the anterior extremity of the superior verm. In this situation it is obvious that it might reasonably be expected to lead to obstruction of the venous circulation of the velum interpositum. Such obstruction should lead to distension of the venous radicles; this, as Carr points out, is usual in the veins seen upon the walls of the lateral ventricles. In none of the cases examined, however, was there any evidence of thrombosis, or even distension of the choroid plexus, though this condition might be expected to be consequent upon venous obstruction.

The third view, which supposes the exudate to be inflammatory, seems most in accord with the appearances found upon the post-mortem table. The main objections to this view are based upon the chemical composition of the fluid, which, it is stated, does not suggest an inflammatory origin. Unfortunately it was not possible to investigate fully the chemical composition of the fluid obtained by lumbar puncture, since the intention of the research was to establish the bacteriology of the disease, and the supply was insufficient for the two purposes.

It does not, however, seem that the analyses of cerebro-spinal fluid hitherto made have been classed according to the stage of the disease at which the fluid was withdrawn. The turbidity of the fluid, the presence of lymph in the lateral ventricles, and the microscopical proof of the presence of cellular elements in several cases were at least

suggestive of inflammatory processes. Possibly even the ventricular fluid may have been secreted by the inflamed pia and arachnoid, and passed upward under tension through the foramen of Magendie.

It is conceivable that, as the condition tends to become chronic, the fluid may cease to contain albumen, and it is only at this stage that the fluid is present in sufficient quantity to enable chemical research to be made.

Further investigations in this direction are necessary before the mechanical theories can be fully rejected.

Dr. Carr suggests a combination of causes: a certain degree of inflammation of the ependyma and membranes, with some obstruction in the velum interpositum. The objections that seemed to discount the venous obstruction theory seem still to hold good; in none of the cases examined was there any engorgement or thrombosis of the choroid plexus. Moreover the presence of fluid in the cortical subarachnoid space, in the basal arachnoid cistern, and in the spinal subarachnoid space, cannot be produced by any obstruction at the great transverse fissure of the brain. Probably, therefore, future investigations will show that there is some fallacy in the objection, based upon chemical analyses, against the inflammatory theory. It may yet be shown that there is a form of leptomeningitis with effusion comparable to pleurisy with effusion or the ascites of tuberculous peritonitis.

Some Observations on the Relationship of the cases here described to Conditions previously described by others.

In the cases in this paper the constant factor has been the presence of the diplococcus intracellularis of Weichselbaum, or an organism closely resembling it, in the cerebro-spinal fluid.

Starting with this insight into their etiology, it has been possible to group together the cases and to demonstrate that they present from their clinical aspect many

points of resemblance. It has further been shown that the signs and symptoms of the disease vary according to its stage. Thus, some have died apparently from the acuteness of the initial disease; others from secondary complications, of which by far the most important is hydrocephalus; while others have passed safely through both these stages and achieved a recovery either complete or incomplete.

It remains now to discuss the relationship of this disease to conditions previously described. Weichselbaum originally isolated the diplococcus which bears his name from certain cases which were considered to be sporadic instances of epidemic cerebro-spinal meningitis. Recently Still and others have isolated a similar organism from cases which clinically resembled what was originally termed by Gee and Barlow the cervical opisthotonos of infants, and has more recently been included under the title of posterior basic meningitis.

It is still a point waiting for decision whether Weichselbaum's diplococcus, as the Germans and Americans hold, is the specific organism of epidemic cerebro-spinal meningitis, or whether, as the French and Italians believe, that disease is due to a modified form of the pneumococcus, or whether different epidemics may be due to different organisms. These points can only be finally decided by the systematic examination of genuine epidemics.

The cases which Weichselbaum investigated were six in number, and were spread over a period of three years (1885—1887); at the same time there were a few other similar cases in Vienna, but there was nothing of the nature of an epidemic. It is true that in the year 1885 there was an epidemic at Mailberg, in Lower Austria, but it was not suggested that this was a possible source of infection. The clinical account of the cases is brief, but they seem to have resembled the cases described by Sanderson as examples of the epidemic form of the disease.

Netter, in describing a typical case of the epidemic variety, speaks of three stages: firstly, that of invasion, lasting from a few hours to three days; secondly, that of reaction, in which the rash appears and the symptoms are ameliorated; and thirdly, that of purulent meningitis. The average duration of the disease, he says, is about twenty days, and, as a rule, if not fatal, the convalescence is long and tedious.

The signs are those of acute cerebro-spinal leptomeningitis, together with those of a more or less acute septicæmia, indicated by a purpuric eruption, the typhoid state, and the enlargement of the spleen.

The cases now reported show no tendency to division into these stages, and present no septicæmic symptoms.

In the circumstances it is impossible to dogmatise; instances are not far to seek of diseases with a different degree of virulence in the sporadic and in the epidemic forms. It may yet be proved beyond question that the epidemic form is due to the diplococcus intracellularis. For the present it is only certain that there is a group of cases occurring with a sporadic distribution in which an acute leptomeningitis is associated with an organism closely resembling, and probably identical with, that which Weichselbaum, in 1887, isolated from cases which apparently presented somewhat similar clinical characteristics.

The best available description of a series of cases of epidemic meningitis is that given by Sanderson in a report to the Local Government Board in 1865, concerning an epidemic then obtaining in the region of the lower Vistula. The disease as seen by him was characterised by a sudden onset, with shivering, headache, and profuse vomiting, followed in a few hours by confusion of thought and pains in the neck, lumbar region, or abdominal wall; delirium rapidly supervened, and with it retraction of the head; vomiting often ceased with the loss of consciousness. In the fatal cases insensibility ensued, and death generally resulted from respiratory failure, appa-

rently due to a spread of the inflammatory process to the medulla. In other cases there was a prolonged period of nervous depression, marked by frequent relapses to the initial symptoms, which ushered in a tedious convalescence. In very few cases consciousness returned early and recovery was rapid.

The temperature as a rule was raised, being seldom below 100°, and generally between 102° and 104°. It was highest during the period of invasion, and rose again with any exacerbation of symptoms. When the disease attacked children, he said that the diagnosis from tuberculous meningitis might be of extreme difficulty. He did not attach great importance to muscular tenderness, and suggested that it occurs as an interlude to the pain, and as a harbinger of convalescence. Labial herpes was apparently of common occurrence, but he only quotes one case in which there was a petechial eruption, and associated with this at the autopsy there was found to be a considerable enlargement of the spleen.

Post-mortem examination showed a purulent leptomeningitis affecting the meninges of the vertex and base of the brain, the upper surface of the cerebellum, and both surfaces of the cord. In three cases which died before the eleventh day of the disease there was no excess of fluid in the ventricles; in the fourth, which died after eighteen days, it is stated that the ventricles were distended with turbid fluid.

These cases, save that they appear to have been somewhat more severe than those now related, present many points of similarity, both in their clinical history and in their morbid anatomy. With regard to age incidence, he shows that children are affected very much more frequently than adults; thus 318 died who were under fourteen years of age, and only 17 above that age, in the period January 2nd to March 31st, 1865, in the district of Berendt.

Though the disease occurred undoubtedly as an epidemic, Sanderson's conclusion was that "no facts were

met with in the course of the inquiry which afforded ground for believing that epidemic meningitis was capable of being communicated by personal intercourse," a statement that is equally true for that occurring in East London in 1901.

Gee and Barlow, in 1878, described and named the condition known as the cervical opisthotonos of infants, and have since continued the investigation of the disease. From the first they suggested a possible relationship to epidemic cerebro-spinal meningitis, but it was not until Still, in 1898, succeeded in isolating an organism which only differed in minor details from that described by Weichselbaum, that the alleged connection became a pathological probability.

There can be no doubt but that many of the cases here described could very reasonably be diagnosed as "posterior basic meningitis;" the point seems to strengthen the credibility of Gee and Barlow's supposition, and also establishes the fact that this disease, though occurring with greater frequency in young children, may occur at least up to the age of thirty-five. The localisation of the purulent exudate to the base of the brain may possibly be explained by the stage of the disease at which the autopsy was performed. In the later stages of the disease it seems to be the rule that the purulent matter remains longest in the basal arachnoid cistern, where at the height of the disease it has existed in greatest quantity.

That the disease may be the cause of acute hydrocephalus is proved by some of the cases quoted; that it may lead to chronic hydrocephalus with enlargement of the skull is probable. Two cases (Dorothy R—, Case 14, and Henry P—, Appendix II) are here given in support of this view, and Dr. Ainley Walker permits me to state that he has isolated Weichselbaum's diplococcus from a case of chronic hydrocephalus which occurred at Guy's Hospital.

Hanshalter[1] has recently reported a case of chronic

[1] 'Les Cliniques Médicales Iconographiques,' p. 127, March, 1902.

hydrocephalus in a child aged six weeks, which was secondary to pneumococcal meningitis; unfortunately, however, the cultural reactions of the organism are not recorded, so that bacteriological certainty is not obtained with regard to the nature of the organism. It does not, therefore, seem improbable to suppose that many cases of chronic hydrocephalus, first becoming manifest after birth, may be not idiopathic, but secondary to an antecedent meningitis.

Bastian, in the last edition of Quain's 'Dictionary of Medicine,' is not inclined to think that this is a common event; it is a point that can only be settled by following up the subsequent history of cases which have been known to suffer from acute leptomeningitis before synostosis of the cranial vault, and from which a definite organism has been isolated.

Conclusions.—Passing these considerations in review, it seems necessary to arrive at the following conclusions:

1. That infection by the diplococcus intracellularis of Weichselbaum has been shown to be associated with a train of symptoms to which have been applied the names epidemic cerebro-spinal meningitis, cervical opisthotonos of infants, and posterior basic meningitis.

2. That these conditions are therefore identical in their etiology, and are probably identical with certain epidemics of cerebro-spinal meningitis which occurred before the introduction of bacteriological methods of diagnosis.

3. That cases of chronic hydrocephalus sometimes are consequent upon this form of acute meningitis.

Table IX.

A comparison of the clinical characters of—

(1) Epidemic cerebro-spinal meningitis as described by Netter in 'Twentieth Century Practice of Medicine.'

(2) Posterior basic meningitis as described by Still in Allchin's 'Medicine' (1901).

(3) The cases reported in this paper.

	Epidemic cerebro-spinal menin'tis (Netter's description).	Posterior basic meningitis (Still).	Cases reported in this paper.
Age	Up to 35 (children especially)	Common in first year, rare after second year	2 months to 35 years.
Evidence of spread by contagion	Nil	Nil	Nil.
Epidemics among persons closely associated	Yes	Nil	Nil.
Onset	Sudden	Sudden	Rapid.
Headache	Present early	Impossible to detect	In older patients.
Vomiting	Present	At onset and often throughout	At onset and commonly in later stages.
Constipation	Common at onset	Not common	Not common.
Rigidity of neck	Common	Constant	In all but one case.
Rigidity of limbs	At times	Common	At times
Kernig's sign	Common (45 out of 50)	Common	13 out of 22.
Temperature	Pyrexia early; 102°	Irregular pyrexia 2 to 3 weeks	Pyrexia early; normal later.
Pulse	Infrequent at onset; accelerated later	Frequent throughout	Frequent throughout.
Respiration	Often periodic	Periodic towards end	Periodic towards end in some cases.
Emaciation	Rapid	Rapid	Rapid.
Mental disturbance	Profound and early	Impossible to detect	In the older cases.
Champing of jaw	At times	Common	Common.
Convulsions	At times	Sometimes at onset; common later	Sometimes early; common later.
Retraction of lids	No mention	Common after 3rd and 4th week	Common in later stages.
Hydrocephalus	Sometimes a sequel	Common sequel	Common sequel.
Photophobia	Common	—	Not common.
Blindness	Sometimes	One third of cases	Common.
Strabismus	Sometimes; varies in different epidemics	Transitory if present	Dissociated movements of eyes common.

	Epidemic cerebro-spinal meningitis (Netter's description).	Posterior basic meningitis (Still).	Cases reported in this paper.
Optic neuritis	No mention	Uncommon	Uncommon.
Rashes	On 3rd day; (*a*) herpes labialis; (*b*) petechiæ; (*c*) rose rash	Rare	Rare.
Stage of improvement with eruption	Present	Not present	Not present.
Joint pains	Common	4 in 40 cases	No instance.
Definite stages	(1) invasion; (2) reaction; (3) stage of purulent meningitis	—	(1) Stage of meningitis; (2) Stage of hydrocephalus.
Duration	Average about 20 days	Up to 3—4 months	3 days to 6 months.
Sequelæ	Generally none; sometimes neuralgia, paralysis, deafness, amaurosis, hydrocephalus, mental troubles	Hydrocephalus; idiocy or imbecility	Hydrocephalus; imbecility; blindness; deafness; spastic rigidity of limbs, etc.
Diagnosis from	Typhoid, typhus, influenza	Tuberculous meningitis; secondary meningitis; head retraction, due to disease of teeth or ears	Tuberculous meningitis; secondary meningitis; typhoid; influenza; cervical caries, or retro-pharyngeal abscess; head retraction, due to disease of ears or inflamed glands secondary to impetigo of scalp.

Historical Survey.

The history of scientific progress in the study of meningeal disease has been so frequently and so well reported, and within so recent a period, that a full and complete account is rendered unnecessary.

Such accounts, however, have, as a rule, treated the subject from one particular aspect alone, and for this reason an attempt has here been made to mass together in one short account a series of references to the chief

discoveries that have marked the progress of this branch of medical science.

Chronological order, so far as was possible, has been maintained, and an attempt has thus been made to show how several diseases, at one time considered distinct, have, with the progress of knowledge, been proved to be related one to the others.

A bibliographical record has been appended giving references to works of which mention has been made, and showing where a more complete account of the literature, which has now become so vast, may be obtained.

In the earliest medical literature no evidence can be found to suggest that inflammation of the coverings of the brain was looked upon as a disease; that the disease then existed may be inferred from descriptions of conditions which, in all probability, represented the secondary effects.

The year 1768 marked the dawn of a new era in the study of cerebral disease. Robert Whytt, of Edinburgh, recorded his memorable observations upon acute hydrocephalus; most, if not all of his cases were due to tuberculous meningitis, but he thought that the determination of the fluid to the ventricles was primary and the cause of the disease. His description of the clinical characteristics of the condition and their division into the three stages form the boundary between chaos and order in the history of intra-cranial inflammatory disease. In conformity with the conception that the effusion was primary and all-important, he employed the title "acute hydrocephalus," a term which continued in general use until 1825, when Senn urged the propriety of substituting the word meningitis.

The work of Whytt was soon followed by that of other investigators. Quin, of Dublin, in 1780 is to be credited with the suggestion that the effusion in the ventricles was secondary, but he went no further than to suggest as a cause a morbid state of the blood in the cerebral vessels.

In the same year Edward Ford suggested, as possible

causes of the dropsy, inflammation of the pia mater, or a tuberculous induration of the brain or cerebellum.

In 1789 Isaac Rand presented a report to the Massachusetts Medical Society on 'Hydrocephalus Internus,' in which he states that "it is probable that the effusion of water into the ventricles of the brain is the effect of inflammation of the meninges." He quotes Whytt, and credits Quin with the recognition of the secondary nature of hydrocephalus, but does not seem to have been familiar with the work of Ford.

Thus at the end of the eighteenth century it seems to have been generally recognised that acute internal hydrocephalus was secondary to some antecedent meningeal or vascular disturbance. At the commencement of the nineteenth century inflammatory conditions of the cerebral meninges came to be recognised as associated with certain symptoms.

Herpin, in 1803, a surgeon with the Army of the Rhine, introduced the term meningitis to express inflammation of the meninges.

The cases that he describes were secondary to fracture of the skull, and consequently unimportant from the point of view of this research. His analysis of symptoms, however, and their contrast with those of what he termed encephalitis, which apparently followed concussion without fracture of the skull, formed a useful basis for those who subsequently investigated the primary form of the disease.

Herpin's seems to have been the first attempt to associate definite symptoms with inflammation of the meninges apart from hydrocephalus. In 1806 there occurred at Geneva an epidemic of a disease with which the physicians of the time were not familiar; it was described by Vieusseux under the title "fièvre cérébrale maligne non-contagieuse." This disease occurred in several members of the same household, but apparently was not communicable by personal contact. It affected chiefly children and young adults, nine tenths of the cases being under thirty years of age, and was not particularly dan-

gerous. The onset was sudden, with headache, vomiting, and delirium, and it might prove fatal in twenty-four hours. In the cases that were not fatal the recovery was rapid and complete, the longest duration being fourteen days.

Unfortunately the report of the post-mortem appearances is unsatisfactory, and in the allusions made to the autopsies there is nothing to suggest that purulent inflammation of the meninges was ever found.

It has, however, been usually accepted that this is the first account of what is now known as epidemic cerebro-spinal meningitis. Mathey gives an account of one autopsy which he conducted for Vieusseux, and describes a gelatinous condition of the meninges which may possibly be taken to indicate the presence of meningitis.

In the same year (1806) Danielson and Mann described an outbreak of a similar disease which occurred in Medfield, Massachusetts, giving the symptoms of the disease and the result of five autopsies. In 1809 a committee, consisting of Drs. Jackson, Warren, and Welch, presented a report concerning this epidemic. They concluded that though it was termed "spotted fever," yet "the petechiæ were secondary and not essential to the disease;" it was a fever of which the "greatest stress fell on the membranes, especially on those within the cranium." They suggested that this inflammation was commonly erysipelatous in nature. The Geneva and the Medfield epidemics drew attention to a disease previously unrecognised, and though there can be no certainty that it was the same as that now known to be caused by the diplococcus intracellularis, yet the investigations that followed laid the foundation for our subsequent knowledge of the subject.

In 1814, Biett, in a Paris thesis on acute idiopathic phrensy, complained that the term was commonly, though wrongfully, applied to inflammation of the arachnoid. Apparently he was the first who recognised idiopathic meningitis as a separate disease, and realised that acute hydrocephalus may follow inflammation of the arachnoid.

In 1815, Golis, in a treatise on acute hydrocephalus, defined dropsy of the head and brain as "a collection of serous, lymphatic, or puriform fluid, or a mixture of these, in the cavities of the cranium or in those of the brain."

This he said might be—

(A) External—that is, either between the scalp and pericranium, or between the pericranium and cranium.

(B) Internal—that is, (*a*) between cranium and dura mater; (*b*) between dura mater and pia mater; (*c*) between pia mater and brain; (*d*) in the cavities of the brain.

(C) Combined, external and internal.

From the description he gives of the cases it seems clear that he dealt with cases not only of tuberculous meningitis and of idiopathic meningitis, but also with cases of secondary meningitis due to fracture of the skull, and possibly also to middle ear disease. In one case in which death resulted from convulsions during an attack of whooping-cough, the record of the autopsy scarcely seems to justify his diagnosis of acute hydrocephalus, seeing that apparently there was no meningitis and no excess of fluid in the cerebral ventricles.

He distinguished two groups of cases which could be recognised by the mode of onset. In the first group the symptoms developed gradually, and apparently closely corresponded to those of what is now recognised as tuberculous meningitis. The second group resembled in the symptoms and mode of onset the posterior basic meningitis of recent years; the disease, he says, commenced suddenly with headache, vomiting, fever, and retraction of the head; and he remarks that the prognosis in this form is better than in the first group; the duration he gives as between thirteen and twenty-one days, and recognises four stages corresponding to the pathological conditions of convulsions and paralysis. In 1809 he had apparently to deal with an epidemic of acute cerebral meningitis amongst the children of Vienna.

Golis, therefore, though he apparently did not clearly differentiate between primary and secondary meningitis,

was able to recognise in his practice cases which corresponded to these groups, and, by drawing a distinction between external hydrocephalus, which in some cases at least was associated with secondary meningitis, and internal hydrocephalus, made an important advance in the study of meningeal disease. Moreover in the matter of internal hydrocephalus he foreshadowed, from clinical experience, a distinction between tuberculous and other forms of acute meningitis, which bacteriological research has since been able to confirm.

Two years later, Coindet, writing about hydrocephalus, includes as causes many other conditions besides meningitis. He gives an account of a case of acute tuberculosis of the lungs, associated with tuberculous meningitis, in a child aged thirty months, but also describes cases of tumours of the brain and pons Varolii, of meningitis associated with otorrhœa, and of acute scarlatinal nephritis with uræmic convulsions, as instances of acute hydrocephalus.

He points out that the condition tends to occur in epidemics:

"Il y a plus grand nombre d'hydrencéphales que dans aucune autre époque pendant les épidémies de fièvres catarrhales. On voit le principe inflammatoire se porter d'emblée sur le cerveau—d'autre fois en le voit se porter d'emblée sur la poitrine—et la quitter pour se jeter sur le cerveau." He noticed that an effusion into the ventricles took some time to form: "Il résulte donc de cet examen cadavérique que dans les morts promptes, il y a peu d'eau, ou que même il n'y en a pas du tout, et qu'il y a beaucoup d'engorgement sanguin; que lorsque la maladie a duré trois semaines ou plus, il y a beaucoup d'eau et moins d'engorgement."

With regard to the cause of the ventricular effusion, he expresses a strong belief that it is due to inflammation of the lining membrane of the ventricles: "L'épanchement dans les ventricules est l'effet et non pas la cause de l'irritation fébrile ou de l'inflammation." The distension

of the ventricles, he thought, caused symptoms of compression or of apoplexy; if not fatal, the effusion, he supposed, might subside completely, or in some instances give rise to chronic hydrocephalus.

Considering these statements, it seems necessary to suppose that he must have observed cases of acute meningitis not due to the bacillus tuberculosis; moreover his deductions seem closely in accord with those reached in the present research.

In agreement with Coindet's views, Senn, in 1825, suggested the use of the term "meningitis" in reference to "acute hydrocephalus," and so began a new chapter in the history of meningitis.

During the next twenty years research was devoted toward the separation of different varieties of meningitis.

In 1827 Guersant recognised a granular form of meningitis, but was not certain that the granules were really tubercles. Papavoine, in 1830, speaks of tuberculous arachnitis and meningitis, but Gerhard, in 1834, was the first to satisfy his contemporaries, by an exhaustive research, that the granules were identical with tubercles.

Guersant, in 1839, in an article on meningitis in a Dictionary of Medicine, makes a distinction between tuberculous meningitis and the non-tuberculous or simple form; the latter, he says, is not uncommon among the newly born, and he quotes Albert (1830) to show that it may possibly occur with an epidemic distribution; neither his work, however, nor Albert's, was based on any extensive investigation of the post-mortem appearances.

In a text-book of diseases of children, published in 1843 by Rilliet and Barthez, is to be found the first accurate description of simple non-tuberculous meningitis, the account of the morbid anatomy being based upon the examination of six cases. They recognised three anatomical forms of meningitis:

1. Inflammation of the pia mater with tuberculous granulations in the meninges.

2. Inflammation of the pia mater without any tuberculous granulations in the meninges, but with general miliary tuberculosis of other organs.

3. Inflammation of the pia mater without any tuberculous granulations in any organ of the body.

The first two groups represent well-known varieties of tuberculous meningitis; the third group, they said, was characterised by pus upon the convexity of the brain, and also at the base, especially near the large vessels, with a slight amount of turbid fluid in the ventricles. In the one case in which the cord was examined there was purulent spinal meningitis.

They further endeavoured to differentiate between idiopathic meningitis and that form which was secondary to an antecedent pneumonia or typhoid or other disease.

Of the idiopathic variety the symptoms were headache occurring early and a disturbance of the mental processes, often resulting in delirium occurring before the third day; vomiting generally occurred at the onset, and sometimes continued until death. The trunk and limbs were rigid and convulsions were common; strabismus was frequently noted, and the patients were often blind. Constipation was only present in two out of the six cases. The duration ranged between one and a half and nine days.

The diagnosis generally had to be made from tuberculous meningitis, and in a table of the chief distinguishing features it is shown that in simple meningitis the onset is more sudden, the headache more intense, the course is more rapid, and the duration much shorter. The pulse is commonly rapid throughout, and does not conform to Whytt's stages.

They held that meningitis in children was, in the vast majority of cases, associated with the tuberculous diathesis, but that a simple form occurred, especially among the newly born. They believed that sometimes epidemics of simple meningitis arose, agreeing in this point with Guersant, and also quoting Albert.

There can be but little doubt that they foreshadowed the work of many subsequent investigators, and arrived at conclusions that have been, after many years, amply confirmed.

Concerning the mechanism by which the effusion in acute hydrocephalus is determined, they put forward a mechanical theory. After discussing the various views that had been previously urged, they point out that if the effusion is due to venous obstruction this must be situated in the course of the veins of Galen, or of the straight sinus, since these are the only veins that drain the ventricles. They say that in some instances they found such obstruction, but that in others no lesion was found in or near these channels, and in consequence suggest that possibly compression occurs as a result of pressure transmitted from below through the cerebellar substance.[1]

In 1844, Gillkrest, an English army surgeon, published an account of an epidemic of meningitis which occurred in that year at Gibraltar. This epidemic was not confined to children, but chiefly affected those between two and eighteen years of age. The onset was sudden, with headache, strabismus, deafness, blindness, etc., and either death occurred on the fifth or sixth day, or marasmus supervened, which might end in death at a later period of the disease, or in recovery after a protracted convalescence. He drew attention to the head retraction and opisthotonos, which was common, and which does not seem to have been noticed by previous authors. He was unable to obtain leave for post-mortem examination upon any children; in the adults he found lymph upon the vertex of the brain, and "the most unequivocal marks of inflammation at the base of the brain." Sometimes he found lymph down the spinal cord; occasionally there was a great excess of fluid in the ventricles. He was familiar with the granular meningitis of Guersant, but

[1] Bastian, in 1867, again put forward the view that the effusion may be the result of thrombosis of the veins of Galen.

did not meet with that condition in any of his cases. Gillkrest undoubtedly had to deal with cases which conformed to the group that Rilliet and Barthez had described under the name of simple meningitis, and his work was the first which gave the evidence of morbid anatomy in support of their view that the disease might occur in an epidemic form. Moreover, seeing that his practice was not confined to children, he was able to show that it might occur in later life.

The next important contribution to the study of meningeal disease was made by Hilton in his lectures on 'Rest and Pain,' where he suggested his famous mechanical theory for the production of internal hydrocephalus. He supposed that owing to a congenital condition, or to previous inflammation in that region, the foramen of Magendie became blocked, so that the draining of the cerebro-spinal fluid from the ventricles was prevented. The probability of the explanation has been discussed elsewhere; it was so specious, and emanated from so high an authority, that it has loomed largely, perhaps too largely, in the writings of subsequent investigators.

Later, in 1865, Burdon Sanderson presented a report to the Privy Council concerning an epidemic of cerebro-spinal meningitis then prevalent in the region of the lower Vistula. This report, containing an accurate account both of the clinical characters and of the morbid anatomy of the disease, with an investigation of its epidemiology, forms the first trustworthy standard to which reference can be made in determining the identity of other outbreaks to which a similar name has been applied. He established an important point in that he showed that "no facts were met with in the course of the inquiry which afforded ground for believing that epidemic meningitis was capable of being communicated by personal intercourse." In times when bacteriology could not be invoked to determine the etiology of a disease, evidence of transmission by contagion was an important point for investigation; meningitis, it is now admitted, may be due to infection by several different

organisms, and it is quite probable that the mode of infection may be different with different varieties. Sanderson showed that one variety of meningitis at least could be epidemic, though not spread easily by contagion; granting similarity in clinical characters and in morbid appearances, evidence concerning the mode of spread must weigh heavily in forming a judgment concerning the nature of two independent outbreaks.

Webber, writing in 1866, in reviewing all the past literature concerning epidemic meningitis, concludes that "it is only epidemic typhus, wherein, from some cause, the cerebro-spinal system is the principal seat of the attack."

The clinical characters of the cases recorded by Sanderson do not suggest any relation to typhus, and the absence of evidence of spread by contagion seems to render such a relationship highly improbable.

Murchison admitted that meningitis might be a complication of typhus; it seems a natural deduction that some of Webber's cases at least may have been diagnosed erroneously, and that they did not belong to the same group as Sanderson's cases, but were due to some other infection, possibly that of typhus.

Since this time, however, epidemic cerebro-spinal meningitis has been recognised as a distinct and independent disease characterised by a definite group of symptoms. It was from cases belonging to this type that Weichselbaum, in 1887, isolated the organism which bears his name, and which is supposed to be the infecting agent.

In 1878 Gee and Barlow published a paper concerning the cervical opisthotonos of infants, but although they suggested its possible relationship with epidemic meningitis, they were unable to find evidence of transmission by contagion, and did not consider that the disease occurred with sufficient frequency to deserve the title of epidemic. In all, they investigated twenty-five cases, the oldest of whom was aged nineteen months; the onset, they said, might be sudden or gradual, and the retraction of the head, which suggested the name, was associated with

rigidity of the limbs and sometimes with epileptiform convulsions. Sometimes enlargement of the head occurred. At the autopsies basic meningitis was found without any evidence of tuberculous infection; in two cases spinal meningitis was present, in one there was internal hydrocephalus, and in five a small effusion into the ventricles.

These cases seem closely allied to the condition described by Rilliet and Barthez as simple meningitis; retraction of the head, however, had not been previously noticed as a prominent feature of the disease, except by Gillkrest in the epidemic at Gibraltar. These cases have more recently been included under the heading of "posterior basic meningitis."

With the development of methods of bacteriological research a new epoch in the study of meningeal disease was inaugurated.

In 1882, Koch, by his discovery of the tubercle bacillus, rendered it possible to be certain concerning the etiology of tubercular meningitis. During the next few years numerous authors described organisms which they considered to be the causal agents of epidemic meningitis, but it was not until 1887 that an accurate description with cultural reactions was given by Weichselbaum of an organism isolated from the cerebro-spinal fluid of certain cases of cerebro-spinal meningitis. Weichselbaum dealt with eight cases, and they occurred during the years 1885, 1886, and 1887.

From two cases he isolated the pneumococcus; from the remaining six he obtained an organism to which he applied the name diplococcus intracellularis meningitidis, or, briefly, meningococcus.

The cases with which Weichselbaum dealt were sporadic, and from the brief clinical report they seem to have conformed to the type investigated by Sanderson on the lower Vistula, and not to the type described in some epidemics where the hæmorrhagic tendency and enlargement of the spleen are so suggestive of a septicæmic condition.

About this time (*i. e.* 1886 and 1887) Netter and Foa

and Ufferedu zzi isolated the pneumococcus from cases diagnosed as epidemic cerebro-spinal meningitis.

Both the pneumococcus and the meningococcus have been frequently isolated by other observers since that time; it is unquestionable that both organisms may give rise to cerebro-spinal meningitis, and it is generally admitted now that they are distinct. It has not, however, been finally settled whether the epidemic form of the disease is always due to infection by the same organism. Differences in the clinical manifestations suggest that all epidemics do not claim the same infective agent.

In 1891 Wynter showed that in cases of tuberculous meningitis the ventricles could be drained by opening the spinal theca in the lumbar region, thus casting doubt upon the truth of Hilton's mechanical theory of the production of hydrocephalus. In the same year Quincke described the process of lumbar puncture, which has since proved to be of such great value in determining the nature of the bacterial infection.

In 1893 Merton demonstrated that hydrocephalus might exist without closure of the foramen of Magendie, and thus supported Wynter's objection to Hilton's theory.

In 1897 Dr. Walter Carr published a long and accurate description of the posterior basic meningitis of infants, entering fully into the clinical aspect and morbid appearances, and discussing at length the pathological conditions which gave rise to the various symptoms. This is the first important paper that deals with this subject since the publication of Gee and Barlow's cases; unfortunately it lacks bacteriological confirmation. This was supplied in the following year by Barlow, Lees, and Still in their article in Allbutt's 'System of Medicine,' in which, in addition to an accurate clinical account of the condition, is recorded Still's observation that an organism closely resembling, if not identical with Weichselbaum's meningococcus, was the infecting agent.

Thus bacteriological evidence was supplied showing the probability of the relationship of posterior basic meningitis

to epidemic meningitis, a relationship which on clinical grounds had been surmised by previous authors.

In the same year (1898) appeared the report of Councilman, Mallory, and Wright, concerning a number of cases of cerebro-spinal meningitis from many of which they had isolated an organism which in its morphological and cultural reactions they considered to be identical with Weichselbaum's diplococcus. The frequency of occurrence of the disease so far transcended the normal that the adjective "epidemic" seemed justifiable. There was no evidence of spread by contagion, and the clinical accounts of the cases show that they were similar to those described by Sanderson, and to those from which Weichselbaum isolated his meningococcus. In this paper 111 cases were reported, and in thirty-nine of these the infecting organism was isolated and cultivated; in most of the other cases the bacteriological diagnosis was based upon the finding of intra-cellular diplococci resembling the meningococcus by microscopic examination of the cerebro-spinal fluid, the nasal secretion, or aural discharge. In the remainder the diagnosis was based upon the clinical characteristics. Since this time, in America and Germany at least, the specificity of the organism has been deemed established.

In 1899, Netter, whose familiarity with the disease is well known, and whose researches have been spread over so long a period, published a long and important article in the 'Twentieth Century Practice of Medicine,' dealing with the disease from all aspects—clinical, pathological, epidemiological, and historical. He claims that in certain epidemics, at least, the pneumococcus is the infecting agent, and seems not quite convinced that the meningococcus is not an attentuated form of the pneumococcus. He does not attempt to draw any clinical distinction between cases infected by the pneumococcus and those due to the meningococcus.

Netter's weighty opinion has carried with him the French and Italian schools of thought, it being generally

held in those countries that the pneumococcus is the organism of epidemic cerebro-spinal meningitis.

It seems scarcely possible to settle this point until an epidemic of a disease whose symptoms resemble those of malignant fever occurs and is submitted to systematic bacteriological investigation.

In 1899 Osler delivered the Cavendish Lecture on cerebro-spinal meningitis; he believed that the meningococcus was the specific organism of epidemic meningitis, but was fully aware of the possibility of infection by the pneumococcus.

In 1901 Thursfield published some cases which confirmed Still's observation that posterior basic meningitis could be produced by an organism resembling the meningococcus; and a few months later Hunter and Nuthall recorded the bacteriology of the cases related in this paper.

In 1902 Hanshalter published a case of chronic hydrocephalus which, from microscopical examination, he concluded was the result of infection of the meninges by the pneumococcus.

In the present paper, basing the diagnosis upon the bacteriological examinations made by Hunter and recorded by him in the 'Lancet,' an attempt has been made to analyse the conditions produced when this organism attacks the meninges. For this purpose, as has been explained, the cases selected are those only in which a complete bacteriological examination was possible.

Infection of the meninges by the diplococcus has thus been shown to produce symptoms suggesting identity with the disease described by Gillkrest, Sanderson, Weichselbaum, Councilman, and many others as epidemic cerebro-spinal fever, and also with the disease hitherto supposed to be limited to childhood, and described by Guersant, Rilliet and Barthez, Gee and Barlow, Carr, Still, Barlow and Lees, and Thursfield, as simple meningitis, cervical opisthotonos, or posterior basic meningitis. Finally, it has been shown that infection by this organism may be one of the causes of chronic hydrocephalus.

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APPENDIX I.

Case 1.—Kate A—, aged 8 months, admitted April 4th, 1901, died April 5th, 1901. The child was admitted under the care of Dr. Gilbart Smith on April 4th, having been ill one day. The child was breast-fed, and previously had been healthy; the illness started with a fit.

There were signs of broncho-pneumonia in the chest, and there were purpuric spots over the body. There was no retraction of the head, and Kernig's sign was not present.

The next day it was noted that the left pupil was larger than the right, and that both reacted to light. There was some squinting with continuous rhythmical up-and-down movement of the eyeballs. There was some rigidity of the neck, but no retraction of the head. Kernig's sign was not present.

She died quite suddenly.

The temperature on admission was 100·9°, and rose to 105·4° just before death.

At the post-mortem examination there was no evidence of tuberculosis; scattered patches of lymph were found at the base, on the vertex, and down the cord. There was an

excess of fluid in the lateral ventricles, which Dr. Hunter reported to contain the diplococcus meningitidis in pure culture.

Case 2.—Sarah H—, aged 7, admitted February 19th, 1901, died February 22nd, 1901. The patient had been quite well until six days before admission under Dr. Schorstein, when she began to complain of headache and nausea, and vomited once. She had been getting thinner during the week, and for the last twenty-four hours she had been very restless, screaming a great deal, and had had incontinence of urine.

The child lay curled up in bed with the limbs flexed and the head retracted; she was very irritable, and when uncovered asked petulantly for the bedclothes.

No signs of disease were found in the thorax or abdomen. There was no otorrhœa, and no sign of injury to the head. There was left internal strabismus. The fundi, except for a little fulness of the veins, appeared normal. Kernig's sign was well marked. Lumbar puncture was performed and several drams of clear fluid withdrawn, and from it the diplococcus intracellularis was isolated. On February 21st some twitching movements of the arms and face followed an ophthalmoscopic examination; later these movements recurred, chiefly in the face and right arm.

The temperature on admission was 103°, and rising, it continued between 104° and 103° until death.

There was no vomiting.

At the post-mortem examination no evidence of tuberculosis was found in any of the organs. On the vertex of the brain the cortical vessels were markedly congested, and there were scattered patches of dry purulent lymph with a considerable excess of clear fluid in the interpeduncular space. The ventricles were filled with clear, slightly blood-stained fluid; the ependyma had not lost its polish. From top to bottom of the cord there were scattered patches of purulent lymph. There were no coarse patho-

logical changes in the substance of the brain or cerebellum.

Case 3.—Harry G—, aged 7, admitted April 29th, died May 5th, 1901. Five days before admission under Dr. Stephen Mackenzie it was noticed that the boy had incontinence of urine, but, seeming apparently well in other respects, he was allowed to go to school as usual. Three days before admission he went to a swimming bath, where he had a fall, which, however, did not seem serious; in the evening he developed a cough. Two days later his mother was bringing him to the hospital on account of the cough, when he had a fit, and he was still in convulsions when he was admitted.

The fits were repeated at short intervals, being epileptiform in character; there was no return to consciousness between the fits. Lumbar puncture was performed at once, and the symptoms seemed to be temporarily relieved; the fluid was under considerable pressure and spurted out of the tube. The meningococcus was found to be present. In the evening there was found marked retraction of the head; there was restlessness but no twitching. The knee-jerks were not obtained. The next day he seemed torpid and the retraction of the head continued, and Kernig's sign was obtained. There was swelling of the optic disc. The stupor continuing, Mr. Openshaw was asked to see the case with a view to surgical interference.

The skull was trephined over the vertex, and continuous irrigation was established from the subarachnoid space to a cannula inserted into the spinal theca in the lumbar region. The patient, however, died eighteen hours after the operation.

Temperature on admission was 100·4°, rising in the evening to 103°, and then falling gradually to 96·5° on April 3rd, when it rose again to 103°. Vomiting was frequent throughout.

At the post-mortem examination there was no evidence of tuberculosis of any organs; the meninges were con-

gested, and lymph was found in the usual positions in cerebro-spinal meningitis.

Case 4.—Joseph B—, aged 4 months, admitted April 18th, 1901, died April 19th, 1901. The child, who had been hand-fed for the past two months, had been wasting for that time; three days before admission under Dr. Gilbart Smith convulsions had commenced, and had been frequently repeated. His mother complained that he screamed almost continually.

On admission there was marked retraction of the head, and Kernig's sign was present. The pupils were equal and reacted to light, and there was no strabismus. The knee-jerks were not obtained.

The pulse-rate was 164 and the respiration rate 64; The temperature was 102·4°, rising to 104·6° just before death, twelve hours after admission. There was no vomiting either before or after admission.

Lumbar puncture was performed, and the diplococcus intracellularis found in the turbid fluid withdrawn.

Seven days before admission the child had been brought to the surgical out-patient department for a small hernia; the notes state that he appeared marasmic, but apparently there was then no sign of meningitis.

At the post-mortem examination there was no evidence of tuberculosis in any of the organs. There was a large amount of purulent lymph at the base of the brain, and scattered patches of lymph on the vertex and down the cord, especially in the region of the lumbar enlargement.

Case 5.—Deborah C—, aged 2, admitted April 20th, 1901, died April 22nd, 1901. On admission under the care of Dr. Gilbart Smith the child had been ill for three days with a cough; there had been no vomiting and no fits. The temperature was 104·6°, the pulse 100, and the respiration rate 40. There were signs of pneumonic consolidation at the base of the left lung. On the following day continuous rhythmic movements of the right arm were

noticed, with twitching of the face, the mouth being drawn to the right. There was deviation of both eyes to the right, but some strabismus was present. The pupils were equal. The neck was rigid, but the head was not retracted. Kernig's sign was not present, and the knee-jerks were not obtained. The movements ceased at times, and then the right arm appeared to be more flaccid than the left.

The temperature fell in the morning after admission to 98°, but rose again to 106° just before death at four a.m. on the morning of the 22nd. At the post-mortem examination there was no evidence of tuberculosis in any of the viscera. There was some broncho-pnemonia in the lungs. The brain was much congested, and there was turbid purulent fluid at the base, with purulent lymph spreading on to the convexity. There was no excess of fluid in the ventricles.

Case 6.—Alfred C—, aged 32, drayman, admitted May 26th, 1901, died June 1st. This man, who was an alcoholic subject, said that he had had inflammation of the lungs six years ago, and pleurisy on the right side for a few days three months before the onset of the present illness. On the morning of May 25th he noticed a pain in the right side of his chest; there was no chill at the onset and no cough.

On admission under the care of Dr. Hadley the liver was found to be enlarged, and pleuritic friction could be heard in the right axilla. There were no symptoms or signs suggestive of meningeal implication.

On May 28th it was noted that the patient was noisy and delirious at night; the signs of pleurisy persisted.

On the 30th the signs of pulmonary trouble had disappeared and the patient seemed better, though he wandered a good deal in his talk.

On the following day (31st) the neck was noted to be very rigid, but there was no retraction of the head; the knee-jerks were not obtained, and Kernig's sign was present. He became semi-comatose, breathing stertorously

and sweating profusely. There was incontinence of urine. In the evening he died.

The temperature was 100·8° on the evening of admission, and fell to 98·4° on May 28th, but after this it rose steadily and reached 104° just before death.

At the post-mortem examination adhesions were found at the apices of both lungs, and recent pleurisy at the right base, but no evidence of tuberculosis or of consolidation of the lungs. The membranes of the brain appeared milky, especially along the course of the vessels. There was an excess of fluid in the ventricles. No pus, or even definite lymph was found.

Case 7.—Alfred L—, aged 19, gasworker, admitted April 7th, 1901, died April 19th, 1901. This man had been at work until the day before admission. On returning home in the evening he had complained of headache and giddiness. He was admitted under Dr. F. J. Smith in a semi-conscious condition, and was very restless, resisting examination. There were no physical signs to suggest the nature of the disease. On the following morning he seemed exceedingly irritable and restless, refusing his food and resenting examination. The eye movements were good in all directions, and the pupils were equal and reacted to light. The knee-jerks were normal. There was some incontinence of urine. There were no signs of thoracic or abdominal disease.

On April 11th he seemed more conscious, recognising his father and speaking rationally. The knee-jerk was readily obtained on the right side, but only just obtained on the left side. The pupils were equal and reacted to light.

On April 12th the right pupil was slightly larger than the left, and both reacted to light; there was some oscillation of the pupils, which was not respiratory. There was slight right external strabismus; there was no retraction of the lids. The neck was markedly rigid, and the arms resisted passive movement to some extent. Kernig's sign

was present. The knee-jerks were both present and equal; on eliciting the plantar reflex, the toes of the left foot moved up, those of the right foot moved down. There was incontinence of urine. Nasal feeding was necessary. Lumbar puncture was performed.

On April 13th he seemed more sensible, and asked for food; in other respects there was not much change.

On April 17th it was noted that he had had diarrhœa with incontinence of urine and fæces for three days. The improvement which had occurred after the lumbar puncture had passed off, and he had relapsed into a semi-conscious condition with retraction of the head.

The pupils were equal and reacted to light; there was no strabismus. Kernig's sign was present. He complained of occipital pain. The temperature ranged between 98·4° and 101·4°, being generally about 100°; just before death it fell to 97°. Diarrhœa was troublesome during the last five days, but he only vomited once while he was in the hospital. The pulse, at first about 80 to 100, rose to 130 per minute towards the end.

At the post-mortem examination no evidence of tuberculosis was found in any of the viscera; all the organs except the brain and cord appeared healthy. The meninges and grey matter were congested; there was a little lymph upon the superior verm of the cerebellum. The posterior surface of the cord was covered with lymph in its entire extent.

Case 8.—Henry R—, aged 35, admitted March 27th, 1901, died April 13th, 1901. Early in November, 1900, began to have aches and pains all over, chiefly in the head, and was feverish; one week later he had violent pain in the head and neck, and some vomiting, chiefly after food. He was treated at home for a fortnight without improvement, and was then sent to the Brook Fever Hospital with the diagnosis of enteric fever. The medical officer of this hospital has kindly provided the following note:

The patient was admitted on December 11th, 1900, with

a history of a severe headache of three weeks' duration. On admission the patient was delirious, and there was pyrexia which persisted for two weeks; later the temperature became normal, with an occasional rise. For the first ten days there was retention of urine; there was marked tendency to constipation. After the first fortnight there was vomiting every two, three, or four days, often without any apparent relation to food.

Widal's test was negative on December 12th, 13th, and 21st. The knee-jerks were present throughout, the pupils were equal and reacted to light, and with accommodation; the fundi were clear.

There was no nystagmus or deviation of the eyes to the side. He was drowsy, helpless, and slept much. He was sent home at the beginning of March, as there was no improvement, and the diagnosis of enteric could not be sustained. His wife thinks that he was worse on discharge than on admission to the fever hospital. He remained at home for three weeks. Seeing that he was getting more helpless, while the headache and vomiting continued, he was brought to the London Hospital on March 27th, and admitted under the care of Dr. Hadley.

On admission he complained of frontal and occipital headache, which was increased on tapping the skull with the finger; he lay still in bed taking no notice of his surroundings, and moaning from time to time. He understood questions, but his answers were unreliable, and showed that he was only anxious to be rid of them.

There was considerable general wasting. No facial, ocular, or other palsies were detected. There was some nystagmus on extreme movements of the eyeballs. The knee-jerks were much increased, especially on the right side; on this side alone ankle-clonus was obtained.

The plantar reflexes were brisk, the toes moving downwards. With the hips flexed to a right angle, the knees could be extended to about 145°. The fundi were normal. There was some hyperæsthesia over the neck and back of the head. There was no photophobia.

There was no evidence of intra-thoracic or abdominal disease. The urine was normal.

The temperature on admission was 100°, and continued to be irregular, ranging from 97° to 101° until his death. There was occasional vomiting throughout; sometimes there was incontinence and sometimes retention of urine; constipation was marked. The irritability continued, and the headache showed occasional signs of intermission.

On April 11th the patient had a right-sided fit, and later in the day had three more. After the last fit he remained stertorous, with divergent strabismus, contracted pupils (reacting to light), and rigidity of the arms and right leg. On April 12th lumbar puncture was performed, three ounces of fluid being drawn off in which the diplococcus meningitidis intracellularis was found to be present. On April 13th he had another fit and died shortly after. The total duration of the illness was over five months.

The post-mortem examination revealed no gross pathological changes except in the cranial cavity. On removing the dura mater there was found to be a small hæmorrhage into the pia mater at the upper part of the right Rolandic area. The convolutions were much flattened. There was a large quantity of clear fluid at the base of the brain, with thickening of the arachnoid. There were adhesions between the prefontal lobes and in both Sylvian fissures. There was a large quantity of clear fluid in the ventricles, and at the lowest part of each posterior cornu there was a tiny mass of green lymph.

Case 9.—Fanny S—, aged 2½, admitted February 14th, 1901, died March 27th, 1901. The child was admitted under Dr. Sansom with the history that she had been ill for two months, the chief symptoms being vomiting and wasting. She was found to be greatly emaciated and very irritable, crying out when she was touched or the bedclothes moved. If undisturbed she seemed to take notice of nothing. The eyes were depressed, and there was retraction of the upper lids showing the sclerotics above

the corneæ. The abdomen was retracted; grinding the teeth was frequent. There was no marked retraction of the head, no rigidity or paralysis of the limbs, and no squint. No signs of disease were found in the heart or lungs; the fundi appeared normal.

For the first three weeks in hospital the temperature remained mainly subnormal, with occasional rises to 99·2° or 99·4°. Vomiting was frequent. The wasting continued.

During the last three weeks the stupor deepened, and the irritability was lost, the child taking no notice when touched. The vomiting ceased, but the emaciation progressed. Some rigidity of the ankle-joints developed, and also some dissociated movements of the eyeballs. There were some slight general convulsions towards the end. The temperature continued subnormal, and at the last was only 96°. Death seemed to be due to inanition. The day before she died she only weighed 1 st. 3 lbs.

On February 26th lumbar puncture was performed, and the diplococcus intracellularis was found in the fluid thus obtained.

At the post-mortem examination there was no evidence of tuberculosis or other disease in any of the viscera. The cerebral convolutions were flattened, and there was a great excess of fluid at the base of the brain, in the ventricles, and in the spinal canal. The cortex was congested and the convolutions were matted together, especially along the Sylvian fissures. No lymph was seen upon the brain, but there was some yellowish lymph all down the spinal cord, especially in the region of the lumbar enlargement.

Case 10.—May P—, aged 8 months, admitted April 13th, 1901, died May 18th, 1901. The illness began ten days before admission under Dr. Percy Kidd with "inward fits," since when the head had been retracted and the child had screamed a great deal. She had been fed on Nestlè's milk. The fontanelle appeared normal, the neck was rigid, but there was no definite retraction of the

head. With the hips flexed the knees could not be extended beyond about 135°. The pupils were equal and reacted to light; there was no retraction of the lids, and no squint. The fundi appeared normal. The temperature was irregular throughout, ranging from 99° to 101·8° for the first ten days, then for a fortnight between 97° and 99·8°, the last ten days being marked by some irregular rises. Vomiting commenced during the second week, and continued to be frequent until the end. The bowels acted freely, but there was no diarrhœa. After a fortnight it was noted that there was occasional retraction of the upper lids, so that the sclerotics were visible above the cornea, and there were also some dissociated movements of the eyeballs, producing an occasional squint. Kernig's sign was present; there were movements as of mastication, and quasipurposive movements of the arms. Examined a day or two before death, the fundi still appeared normal. Lumbar puncture was performed on April 2nd, and a considerable quantity of fluid removed, in which the diplococcus meningitidis was found. The symptoms seemed to be relieved for a time by the puncture; the operation was repeated on May 13th, but no fluid was withdrawn. Ultimately the child died of exhaustion.

At the post-mortem examination it was found that the convolutions were much flattened and the ventricles enormously distended with fluid, which was straw-coloured and contained a few flakes of lymph. The choroid plexuses were firm and infiltrated. There was a large quantity of fluid within the spinal dura mater. There was no evidence of tuberculosis.

Case 11.—Herbert C—, aged 5 years, admitted July 19th, 1901, died September 5th, 1901. This boy had been subject to attacks of headache all his life. Six weeks before admission to the hospital under Dr. Stephen Mackenzie he had suffered from measles. Three weeks later he had complained of pain in his neck, and five days before he was admitted his neck had become stiff. There

was no history of tuberculosis in the family. On admission there was some rigidity of the neck, but no retraction of the head; the cervical glands were a little enlarged, but there was no evidence of any throat trouble. The limbs were not rigid, Kernig's sign was not present, there was no retraction of the upper lids, and no strabismus. The temperature for the first four days was raised (99° to 101°), and the child was drowsy. There was no vomiting. On July 30th the child was allowed to go to a convalescent home. On August 25th diarrhœa and vomiting commenced, and he was sent back to the hospital. He then complained of pain in the head, and there was some rigidity of the neck; he was drowsy, and there was incontinence of urine. Kernig's sign was obtained, and the retinal vessels appeared full, while the margins of the disc were blurred. There was no evidence of heart or lung disease. On September 2nd the child appeared to be much worse; he seemed to be both blind and deaf so far as could be gathered from endeavouring to get him to take notice of light or sound. The pupils were dilated equally, and did not react to light; the retinal vessels were full, but not tortuous; the margins of the discs were blurred, but no definite swelling could be determined; no hæmorrhages were seen. Kernig's sign was present, and there was some rigidity of both arms and legs, with rigidity of the neck.

Lumbar puncture was performed on September 3rd, and Dr. Hunter reported the presence of the diplococcus intracellularis in pure culture.

The temperature was normal on admission, and gradually rose to about 100° to 101° just before death on September 5th.

There was no post-mortem examination.

Case 12.—Winifred H—, aged 3, admitted July 20th, 1901, died August 9th, 1901. The illness commenced with measles one month before admission; a fortnight later, July 8th, she began to complain of pain in the head and legs, with occasional vomiting. Thoughout this fortnight

the head was retracted, and the temperature ranged between 100° and 102°. There was no history of tuberculosis in the family.

On admission under Dr. Sansom there was marked rigidity of the neck, with retraction of the head; there was no strabismus, and no retraction of the upper lids. The child was very fretful and resented interference; the legs were kept flexed, and attempts to straighten them caused screaming, though the straightening could be accomplished. She complained a good deal of headache; she took her food well and did not appear very wasted. Though apparently not blind, she failed to recognise her mother. There was no evidence of heart or lung disease. The child continued in much the same condition until August 7th (nineteen days), when she developed cancrum oris, from which she died on August 9th. During this time the temperature ranged between 99° and 102·4°, and the weight gradually fell from 1 st. 10 lbs. 3 oz. to 1 st. 8 lbs. 11 oz. There was no vomiting in the hospital. Lumbar puncture was performed on July 31st, and Dr. Hunter reported the presence of Weichselbaum's diplococcus. Post-mortem examination was not allowed.

Case 13.—William H—, aged 1½, admitted April 24th, discharged June 28th, 1901. The child was admitted on April 24th, under the care of Dr. Sansom, with well-marked signs of rickets and a temperature of 103°.

There were no signs of thoracic disease; the liver and spleen were enlarged. He was very irritable on examination; the epiphyses of the long bones were enlarged, and there was a well-marked rickety rosary. The fontanelle was patent, but did not bulge; Kernig's sign was present. On April 28th lumbar puncture was performed, and one and a half test-tubes of fluid withdrawn, which was found to contain the diplococcus meningitidis. On May 1st slight retraction of the head was noticed. On May 8th the child had an attack of convulsions lasting about twenty minutes, in which he became cyanosed, and there was

marked retraction of the head. These attacks were repeated three or four times a day for a week.

In the intervals there were restless movements of the head, eyes, and frontal muscles, but no strabismus was noticed. Kernig's sign was not present; there was no rigidity of the arms, legs, or neck, and no retraction of the upper lids, though at intervals the eyes became "staring." The temperature was irregular, ranging between normal and 104° for eight weeks, and then became normal. Vomiting occurred at intervals, being most marked when the temperature was high.

An epidemic of measles breaking out in the ward, and the child seeming much better, he was discharged on June 28th.

On July 16th he was again admitted to the hospital, having been ill for a week with cough and shortness of breath; there were signs of general broncho-pneumonia and evidence of measles. There was some retraction of the head.

The temperature was 102°. He died on July 20th from exhaustion. There was no post-mortem examination.

Case 14.—Dorothy R—, aged 8 weeks, admitted June 1st, discharged June 10th, 1901. The child was brought to Dr. Hutchison's out-patients' on May 1st, suffering from hydrocephalus. Her mother stated that the head seemed natural at birth, but that it had been increasing in size during the last week. The child had been fed at the breast, and both mother and father were healthy. Her mother had lost one child from bronchitis and convulsions, and had three other children living, who, however, were subject to bronchitis.

The girth of the head was then 17 inches; on May 25th, when next seen, there had been no vomiting or convulsions, but the girth of the head was 18 inches. On June 1st the child was admitted to the hospital under the care of Dr. Sansom. The horizontal girth of the head was then 18¼ inches, and all the bones of vertex were

widely separated. The eyeballs were depressed, and the upper lids were occasionally retracted; there was no strabismus. The limbs showed slight rigidity, but could be straightened; Kernig's sign was not present. The temperature was normal. The optic discs appeared blurred, and it seemed probable that the child was blind.

On June 4th lumbar puncture was performed, and Dr. Hunter reported the diplococcus meningitidis to be present.

At the end of a week the child seemed to be getting worse, and vomiting commenced; the mother's desire to take the child home was therefore acceded to.

On June 18th the child was seen at home, and still seemed very ill; there was slight strabismus, some retraction of the head and some rigidity of the limbs, and Kernig's sign was present.

On July 13th she was again seen in the out-patient department, and had greatly improved. The hydrocephalus remained stationary, but there were no signs of meningitis.

On the 19th of April, 1902, she was again seen, and presented the typical appearance of chronic hydrocephalus. The horizontal girth of the head was then $20\frac{3}{4}$ inches, and the measurement between the auditory meatus $13\frac{3}{4}$ inches.

Case 15.—Solomon B—, aged 6 months, admitted April 4th, discharged June 27th, 1901. On admission under Dr. Gilbart Smith it was stated that the child had been ill for three weeks, starting with convulsions, followed by bronchitis and pneumonia, and that during the last few days he had vomited occasionally and had been convulsed. The mother thought that the child had been blind for four days. He was breast-fed, but had had some cow's milk in addition. There was marked retraction of the head, an occasional squint, and intermittent elevation of the upper lids; Kernig's sign was well marked. The fontanelle was patent and bulged.

Lumbar puncture was performed, and a large test-tube

was half filled with clear fluid, in which no micro-organisms were found. The child continued in much the same condition, with marked cervical opisthotonos, Kernig's sign, occasional retraction of upper lids, and strabismus. The temperature remained normal, and vomiting was infrequent. On April 9th lumbar puncture was again performed, two test-tubes full of fluid being removed. In this micro-organisms were found, but no diplococci. Lumbar puncture was repeated on May 17th and on May 27th, 2 oz. of fluid being removed on each occasion, and on June 8th 3½ oz. were taken away. On June 1st it was noted that there was very slight, if any, rigidity of the neck and limbs.

The child seemed happy and smiling; as far as could be determined he was blind. The movements of the hands were rather jerky, and there was still occasional retraction of the upper lids, so that the sclerotics showed above the corneæ. The discs were clear. The child continued in much the same condition, apparently suffering from internal hydrocephalus, which threatened to be chronic when he was sent home owing to the occurrence of scarlet fever in the ward. For the last four weeks before his discharge the temperature had been quite normal, and vomiting had only occurred very seldom.

Case 16.—Etta S—, aged 1½, admitted May 3rd, discharged May 23rd, 1901. The illness began with convulsions, seven weeks before admission under Dr. Warner. The bowels had been constipated, all food had been vomited, and there seemed to be abdominal pain. The heart, lungs, and abdomen presented no signs of disease. There was very marked retraction of the head, some rigidity of the right arm, but no Kernig's sign. There was no photophobia, no squint, no retraction of the upper lids, no movements of the lower jaw. The fontanelle was closed. The discs appeared normal. The temperature, generally subnormal, occasionally rose for a short time; the highest rise was to 102°. Vomiting occurred only occasionally.

On May 15th the child was found sitting up with no retraction of the head or stiffness of the neck, no rigidity of the arms or legs, and apparently completely recovered.

On admission, lumbar puncture was performed, and the diplococcus intracellularis meningitidis was found to be present in pure culture. The child was discharged cured on May 23rd.

Case 17.—Ellen T—, aged 3 months, admitted March 12th, discharged June 28th, 1901. This child was admitted under the care of Dr. Gilbart Smith on March 12th, with a history that for the past five weeks she had been wasting and that she had had a cough; during the week previous to admission she had had convulsions in her sleep, and for the last few days she had vomited after her food. She had been fed on milk and barley-water.

On admission the child was found to be extremely emaciated, but there was no evidence of thoracic or abdominal disease. There was slight retraction of the head, and Kernig's sign was present; the pupils were equal and reacted to light.

On March 14th lumbar puncture was performed, and in the fluid obtained the diplococcus meningitidis was reported to be present. There was also some convergent strabismus.

On March 15th some œdema appeared over the back of the sacrum and extended to the angles of the scapulæ.

On March 22nd the child appeared much wasted, but was not particularly irritable; she took her food well. With the hips flexed to a right angle with the trunk, the knees could not be extended beyond 135°. There was no strabismus; no retraction of the upper lids, and the fontanelle appeared natural.

On April 9th there was found to be some retraction of the head, and occasional retraction of the upper lids was noticed for the first time.

On April 12th lumbar puncture was again performed, and the diplococcus was reported to be present.

On April 26th the child was better and had gained weight; there was occasional retraction of the upper lids; Kernig's sign was present upon the right side, but not upon the left.

On May 6th there was no retraction of the head, but marked occasional retraction of the upper lids. Kernig's sign was not present on either side.

The temperature throughout was subnormal. For the first four days there was occasional vomiting, but subsequently it only occurred at very infrequent intervals. On discharge the child seemed to have recovered almost completely; it seemed probable that the child was neither deaf nor blind. The weight, which shortly after admission sank to 6 lbs. 14 oz., rose to 8 lbs. 9 oz. on discharge. The intermittent retraction of the upper lids, however, persisted.

On July 6th she was admitted for a few days for convulsions, which, however, were not repeated in the hospital; no evidence of disease was then found.

Case 18.—Esther L—, aged 6 months, admitted April 20th, discharged June 27th, 1901. The child on admission under Dr. Gilbart Smith had been ill for five weeks, being very feverish and fretful and vomiting occasionally. The head had been retracted throughout.

On admission the head was retracted and the fontanelle bulged; Kernig's sign was not present. The knee-jerks were equal and easily obtained. The pupils were equal and reacted to light; there was no squint. The fundi showed no pathological appearances. There was no evidence of thoracic or abdominal disease.

Lumbar puncture was performed on April 21st, and the fluid was reported to contain the meningococcus. On the following morning it was found that the fontanelle was level and pulsated. The head was markedly retracted, but there was no rigidity of the arms or legs, and Kernig's sign was not present. There was no squint; the pupils were equal and reacted to light. The eyes were generally

kept open, and there was no photophobia. Occasionally the upper lids were slightly retracted, but not sufficiently to show the sclerotics above the corneæ. The fundi appeared normal. The child took her food well from the bottle, though she had been breast-fed until admission.

On April 26th the child was lying quietly, apparently taking notice of her surroundings. The upper lids were occasionally retracted so as to allow a band of sclerotic to appear above the corneæ; there were occasional dissociated movements of the eyeballs, but there was no permanent squint. The head was slightly retracted. There was no rigidity of the arms and legs, and Kernig's sign was not present. There were no continued movements of the lower jaw.

On May 8th the neck was still rigid, but there was no retraction of the head. The occasional retraction of the upper lids was still present. There was no squint and no rigidity of the legs; the left arm was markedly rigid, and kept closely apposed to the chest.

On June 1st there was but slight rigidity of the neck, and no retraction of the head. The occasional retraction of the upper lids persisted, but there was no squint or dissociated movement of the eyes, and no rigidity of the limbs. The child seemed to be gaining ground, but still had occasional attacks of fever associated with vomiting and malaise. The temperature at first remained at about 99° or 100°, with an occasional rise to 101° or 102°, but the rises became more infrequent, and after about the seventh week in hospital the temperature remained near the normal. Vomiting occurred at intervals throughout the child's stay in hospital, but was never very frequent. She was discharged almost recovered from the meningitis on June 27th owing to an outbreak of scarlet fever in the ward. The weight on admission was 10 lbs. 11 oz., and fell to 9 lbs. 11 oz. during the fourth week, when it began to rise again uninterruptedly, and was 11 lbs. just before discharge.

On July 16th the child was readmitted; she had had

some diarrhœa, but the condition otherwise was not much changed. The head was slightly retracted; there was no rigidity of the arms or legs, and Kernig's sign was not present. There was no squint, but the upper lids were occasionally retracted. The child seemed happy, and the spontaneous movements of the limbs appeared natural; no evidence of cerebral defect could be discovered.

Case 19.—George P—, aged 5, admitted June 8th, discharged July 4th, 1901. The child on admission under Dr. Sansom had been ill for four or five months; for the past two months his walking had been unsteady, and he had frequently fallen and hurt himself. He had had abdominal pain, and vomited every morning about a pint of watery fluid. His mother and father both died of phthisis about two years ago; there were two other children apparently quite healthy.

On admission the child appeared well nourished and happy, but seemed a little foolish. The horizontal girth of the head was 22½ inches. The heart rhythm was irregular, the pulse frequency increasing during inspiration. The lungs were free. There was alternating strabismus, but no nystagmus. The left pupil was larger than the right, and both reacted to light and with accommodation. There was no apparent blindness or deafness; the tongue was protruded in the middle line, and there was no facial paralysis or speech defect. There was marked double optic neuritis.

During examination he was seized with a sudden attack of pain referred behind the left ear, which made him weep copiously. The gait was very staggering, but there was no marked tendency to fall in one direction. The knee-jerks were obtained with great difficulty; the plantar reflex was of the flexor type on the right side, and was not obtained upon the left side. The right cremasteric reflex was present, but not the left. Later the knee-jerks were not obtained. Kernig's sign was not present; in fact, there seemed evidence of some hypotomus in the legs.

On June 10th lumbar puncture was performed, and Dr. Hunter reported the presence of Weichselbaum's diplococcus in the fluid.

On June 12th the boy vomited a round worm. There was no headache, but the vomiting was frequent; the swelling of the optic discs continued, and there was a definite squint. There was no rigidity of the limbs or neck.

On June 17th there had been no vomiting since the 12th, the squint persisted, and the patient complained of pain behind the left ear.

On June 24th the temperature, which had been normal, rose to 100°, and on the 27th a definite measles rash appeared. The measles ran a normal course, and on July 4th the boy was discharged apparently relieved of all his symptoms.

Case 20.—John E—, aged 1½ years, admitted July 6th, 1901, discharged August 30th, 1901. The illness began on September 29th with diarrhœa and vomiting. He was brought to the out-patient department on July 2nd, and was then found to have some bronchitis; he was treated as an out-patient until July 6th, when, as no improvement was manifested, he was admitted under Dr. Schorstein.

There was no history of tuberculosis in the family.

On admission the temperature was 100·2°, and there was some general bronchitis. The diarrhœa was urgent, but there was no vomiting during the first week. Two days later there was noticed marked retraction of the head. This continued, and on July 12th the child began to vomit. On July 15th lumbar puncture was performed, and Dr. Hunter reported the fluid to contain the diplococcus intracellularis in pure culture. On July 19th the child was drowsy and apathetic; there was some rigidity of the neck, but no retraction of the head. The pupils were equal and reacted to light; apparently the patient was not blind. There was no squint, no retraction of the upper lids, Kernig's sign was not present, and the limbs

were not rigid. Vomiting continued at intervals from July 12th to July 21st, and then became infrequent. The temperature during this time rose to 100° each evening, but subsequently was normal.

From this time the child progressed satisfactorily, and on discharge seemed to have completely recovered. The fundi were examined on several occasions, and were never found to present any pathological appearances.

Case 21.—Albert P—, aged 3 years, admitted September 5th, 1901, discharged October 11th, 1901. The illness commenced on September 1st with vomiting and "screaming" fits, which were followed by stupor. There was a history of tuberculosis in the mother's family, and one other child had meningitis, from which she had recovered (Maria P—, in Great Ormond Street, in March, 1893).

On admission under Dr. Warner the child seemed stupid; he was very irritable, and screamed when disturbed. There was marked head retraction, and an intermittent strabismus of the right eye. On September 11th the head retraction persisted, and Kernig's sign was present in both legs, but there was no other rigidity of the limbs. There was no sign of thoracic or abdominal disease. He could see and hear, and understood questions. He complained of pain at the back of the head. There was still an intermittent strabismus. On September 14th lumbar puncture was performed, but no fluid was obtained; later the operation was repeated with success, and 2½ ounces of fluid obtained. Dr. Hunter reported the presence of the diplococcus intracellularis in pure culture. On September 21st the retraction of the head continued, but the child seemed better. On October 7th there was no retraction of the head, but some slight rigidity of the neck. He was not very bright, but otherwise seemed quite well. On Nov. 2nd he was discharged apparently completely recovered. For the first eighteen days the temperature was irregular, ranging from 98° to 101·4°, but subsequently was about normal. Vomiting was never

very frequent, but occurred at intervals during the first four weeks.

Case 22.—John C—, aged 3 years, admitted October 13th, 1901, discharged November 4th, 1901. Three weeks before admission under Dr. Gilbart Smith the child fell out of a barrow and hurt the back of his head. Since this accident he had been irritable and refused his food, becoming delirious at night. For the past week there had been diarrhœa, but there had been no vomiting. There were dissociated movements of the eyeballs, but no definite rigidity of the neck or retraction of the head. Kernig's sign was not obtained. Lumbar puncture was performed, and six drachms of fluid removed, in which Dr. Hunter found the diplococcus meningitidis. The temperature on admission was 100°, but subsequently, with the exception of one evening, was normal or subnormal. Vomiting occurred occasionally for the first eight days, but not subsequently.

For nearly three weeks the child uttered no sound, but apparently could both see and hear. He was discharged on November 4th to attend as a ward out-patient, being still very irritable but able to speak. Since his discharge he has gradually improved, and is now apparently restored to his normal health.

Case 23.—Elizabeth S—, aged 6, admitted March 8th, 1901, died March 13th, 1901. The child was brought to the out-patient department on March 5th, with the history that she had fallen downstairs ten days before and had hurt her back. For the last five days there had been headache and vomiting. The temperature was 101°, but no physical signs of disease could be discovered; she was sent home to bed, to be brought up again on the following day, when no change in the condition was found.

On the morning of the 8th she had a fit, was brought to the hospital, and admitted under Dr. Warner.

There seemed to be no evidence of thoracic or abdo-

minal disease; the temperature was 99·6°. She was unconscious, and lay still save for a rhythmic movement of left hand and forearm, and occasional movement of the jaw as if in mastication. The pupils were widely dilated and fixed, and there was no photophobia. There was slight lateral nystagmus and slight left internal strabismus. There was no rigidity of the neck, but Kernig's sign was present. There was incontinence of urine. Knee-jerks were not obtained.

In the evening lumbar puncture was performed, and about two ounces of clear fluid removed, which proved to contain the meningococcus. On March 11th it was noted that there was slight ptosis of the right lid, and some dissociated movements of the eyeballs, with right internal strabismus. Occasionally she called for her mother, but did not seem to recognise her. There were no twitchings and no marked rigidities. The right pupil was dilated and fixed; the left pupil was smaller and reacted to light. Incontinence persisted. On March 13th there was some rigidity of the neck, but Kernig's sign was not present. Wasting had been very rapid. An ulcer had developed upon the right cornea. Dissociated movements of the eyeballs were noted. The discs were examined several times, but no definite changes were noted. The temperature ranged from 99° to 101° until just before death, when it rose to 102·4°. There was no vomiting while she was in the hospital.

The urine once contained a trace of albumen, but, tested again, it was normal.

Post-mortem examination.—A caseating bronchial gland was found, from which tuberculous infection had spread to visceral pericardium; there was, however, no acute pericarditis.

No evidence of tuberculosis could be found elsewhere.

There was some excess of fluid at the base of the brain and in the spinal theca and in the ventricles; also some purulent lymph was found in the interpeduncular space, on the under surface of the cerebellum, and down the spinal cord.

The brain substance and the pia mater appeared congested.

Case 24.—Bella C—, aged 1 month, admitted and died March 11th, 1901. A seven-months child, one month old, was admitted under Dr. Percy Kidd on March 11th, with the history that she had been suffering from convulsions for one day. There was slight retraction of the head, but no strabismus; the convulsions continued, affecting both arms and legs, and shortly after admission she died.

At the post-mortem examination all the organs appeared healthy except the brain, which showed acute suppurative vertical meningitis. The diplococcus intracellularis and pyogenic organisms were found by Dr. Hunter to be present in the lymph.

Case 25.—Michael G—, aged 4 months, admitted April 4th, 1901, died April 10th, 1901. The child was breast-fed, and was quite well until April 2nd, when he had a fit; on the 3rd there were nine fits, and on the 4th there had been six before admission to the hospital under Dr. Gilbart Smith. During the day of admission there were seven more fits, but they then ceased. The fits consisted of general clonic convulsions, the pupils being dilated and immovable, and the eyes turned to the left. In the intervals between the fits the pupils were equal and contracted, and reacted to light. There was no retraction of the head or rigidity of the body, and Kernig's sign was not present; the temperature ranged between 97° and 99°, and there were no physical signs of thoracic or abdominal disease.

On April 5th the child appeared perfectly well, and continued so; on April 7th he was allowed to be taken home, so that the mother might resume nursing.

On April 9th he was again brought to the hospital, because for thirty hours he had taken nothing; there had been no fits in the interval. The child took the bottle

well in the hospital, and the notes state that there was no bulging of the fontanelle, no squint, and no changes in the fundi. The pupils were large, equal, and reacted to light. The neck was slightly rigid, but there was no retraction of the head; the knee-joints could not be completely extended with the hips flexed, but there was no definite Kernig's sign. The knee-jerks and plantar reflexes were present. No signs of thoracic or abdominal disease could be found. The temperature was 98°. On the following day the temperature rose to 100°, and the child became convulsed, and died.

At the post-mortem no naked-eye pathological appearances were found. There was no evidence of meningitis, and no excess of cerebro-spinal fluid. The diplococcus intracellularis and pyogenic organisms were found in the fluid by Dr. Hunter.

Case 26.—Samuel G—, aged 15 years, tailor. This boy had been ill in bed for three weeks before admission under Dr. Gilbart Smith. The illness commenced with a violent headache, which had persisted; there had been no vomiting; his mind had been clear until the last twenty-four hours, during which he had been delirious and had not recognised his relatives. He had been deaf for five years, and had had double otorrhœa for that time until the onset of the present illness, when the discharge ceased.

On admission he screamed when touched anywhere, and lay with his legs drawn up, and Kernig's sign was present. The neck was rigid, but there was no retraction of the head. The pupils were equal, and reacted to light. There was no otorrhœa, but both membranes were perforated; there was no œdema or tenderness over the mastoids; there was no squint. The knee-jerks were present and equal. Both fundi appeared normal. There was retention of urine.

On April 4th paresis of the right side of the face and of the right arm was noticed; the condition otherwise was unchanged, save that he seemed more drowsy. He was

then transferred to the surgeon. The temperature for these three days was about 99·4°, and there was no vomiting. Mastoidectomy upon the left side was performed, but no pus was found; a trephine hole was made over the left temporo-sphenoidal lobe and this region of the brain, and later the cerebellum was explored. The membranes bulged into the trephine hole, but no pus was found on further exploration. On the following day he seemed slightly better; the facial paresis was less marked; the temperature, however, rose to 101°. On the third day the left pupil was noticed to be larger than the right, and there was some ptosis on the left side, and a little difficulty in swallowing. These signs increased, and complete paralysis of the palate developed. During the last twenty-four hours the breathing became periodic. Death was somewhat sudden.

At the post-mortem examination there was found to be some broncho-pneumonia in the left lung. The cerebral ventricles were distended and the convolutions flattened; there was a large quantity of clear fluid at the base of the brain.

Dr. Hunter reported the fluid to contain the meningococcus in large quantities, and also pyogenic organisms. There was no lymph seen, and no evidence of tuberculosis. The site of the mastoidectomy was healthy, and also the petrous bones. Both middle ears contained pus.

Case 27.—Isaac M—, aged 19 years, carman, admitted May 7th, 1901, died May 14th, 1901. This man had been quite well until the day before admission under Dr. Gilbart Smith, when he had been suddenly attacked by headache, drowsiness, vomiting, and diarrhœa, with stiffness of jaws.

On admission he was in a semi-comatose, irritable state, resenting interference forcibly, and making no reply to questions; he apparently, however, did not resent a hard pin-prick any more than a light touch. He lay upon his left side, huddled up, and did not move unless disturbed. The neck was rigid and the head a little retracted; the

pupils were dilated and fixed. There was no strabismus. The fundi showed no pathological changes. There were no signs of paralysis of face or limb, no signs of mastoiditis, no tenderness over the occiput or spine, and no evidence of thoracic or abdominal disease. The legs were rigid, but Kernig's sign was not obtained; the knee-jerks were obtained on both sides, and Babinski's phenomenon was present. The temperature was 100·4°. On May 8th a catheter was passed in consequence of retention of urine, and 20 oz. were withdrawn. He became very violent and needed shackling, but quieted down and slept for half an hour after an injection of morphia; he was very restless afterwards, constantly calling for his friends. He took his milk fairly well through the day, but had incontinence of urine. In the evening the pupils were equal and reacted to light. He passed a fairly quiet night, but became noisy on the morning of the 9th; there was then a divergent strabismus, but in other respects his condition was unchanged. He slept under the influence of morphia. On May 10th the pupils were equal and reacted to light, but there was no fear reflex, and it seemed probable that he was blind. There was considerable retraction of the head, and attempts at flexion caused him to call out: "Oh! my head and neck." There was no strabismus, and the fundi appeared normal. A patch of herpes appeared upon the right cheek; there was marked distension of the abdomen; incontinence of fæces as well as urine set in. In the evening signs of congestion of the bases of the lungs developed. On May 11th the condition of noisy delirium still persisted; the knee-jerks were not obtained, and Kernig's sign was present. In other respects his condition remained almost unchanged. On the 12th and 13th he gradually became quieter, and developed some signs of consolidation at the base of the right lung. The temperature on admission was 100·2°, and rose to 103° on the following morning; then it fell to 99° for three days, rising to 103° on May 11th, gradually falling to 100° on the morning of May 14th, just before death. While in

hospital he vomited three times, and except just at first there was complete incontinence of both urine and fæces.

At the post-mortem examination there was found to be pneumonia at both apices in a stage approaching grey hepatisation ; there was also some consolidation at the left base. The brain showed some streaks of purulent matter over the vertex ; the membranes at the base were milky, and there was some definite pus on the under surface of the cerebellum. There was no excess of fluid in the ventricles, except that there was some pus in the fourth ventricle. The brain substance, though, appeared pinker than normal. Purulent inflammation extended down the whole length of the cord.

Dr. Hunter reported the presence of the diplococcus intracellularis meningitidis and of the pneumococcus in the cerebro-spinal fluid.

Case 28.—Jessie B—, aged 1 year and 3 months, admitted May 7th, 1901, died May 23rd, 1901. The child had been bottle-fed since birth, and had been healthy, save for one fit when three months old, until a week before admission under Dr. Gilbart Smith, when she had another fit. She had screamed a great deal since, and though she had had no recurrence of the convulsions the mother said that "she went stiff when touched."

On admission the temperature was 100°, and it rose to 102° on the following morning. There were signs of general bronchitis. The fontanelle was patent and bulging ; there was retraction of the head. The pupils were equal and reacted to light; it was doubtful if there was some strabismus. There was no retraction of the upper lids. The knee-jerks were not obtained, and Kernig's sign was not present. Lumbar puncture was performed, and the fluid was reported to contain the pneumococcus and diplococcus intracellularis. On May 9th, after lumbar puncture, the fontanelle was noticed to pulsate ; the head was markedly retracted, but there was no rigidity of the limbs, and Kernig's sign was not

present. The bronchitis persisted and the cough was frequent; the child yawned often; there was no squint, and no retraction of the upper lids. On May 11th the fontanelle was found to be bulging but pulsating; the head was markedly retracted, but there was no rigidity of the limbs, and Kernig's sign was not present. There was no squint and no retraction of the upper lids. The optic discs appeared blurred on admission, and subsequent notes reassert this, but there was no definite evidence of optic neuritis. The temperature continued between 100° and 102°, with only two intermissions, until forty-eight hours before death, when it fell to 99° and continued there. For the last nine days of life vomiting was frequent, but had not occurred in the earlier part of the illness.

The post-mortem examination showed general bronchopneumonia. There was purulent meningitis limited to the basal subarachnoid cistern and under surface of cerebellum, and spreading all down the cord. There was no excess of fluid in the ventricles.

Case 29.—Abraham D—, aged 2 years and 10 months, was admitted to the London Hospital on January 30th, 1901, under the care of Dr. Stephen Mackenzie, with the history that he had been ill for two months with fever, irritability, and attacks of cramp, with marked wasting. He had been fed on milk and beef tea since the commencement of the illness; there had been no vomiting; the bowels had only acted after medicine. The only previous illness was measles eighteen months before. The child was found to be irritable, with a puny cry; the head was rigidly retracted. There was no discharge from the ears; the pupils were dilated, equal, and reacted to light. There was no optic neuritis. The knee-jerks were increased; the plantar reflexes were normal, the toes moving down. There was no rigidity of the legs either with the hips extended or flexed to a right angle. Everything was passed under him. He continued in the same condition until February 10th, when vomiting set in and

continued almost daily until his death. On February 21st some internal strabismus was noted, and occasional retraction of the upper lids, showing the sclerotic above the cornea. The retraction of the head continued; there was no Kernig's sign. Definite optic neurititis developed. Wasting became more obvious. On February 26th lumbar puncture was performed. The fluid obtained proved to contain diplococci meningitidis and probably bacillus influenzæ. The wasting increased, and he died from exhaustion on March 4th.

On admission the child weighed 19 lbs. 6 oz.; at first he gained a few ounces, but when the vomiting commenced he began to lose weight, and a week before death weighed only 18½ lbs. The duration of the illness was three months. The temperature, as a rule, was subnormal; once, three days before death, it rose for a few hours to 102·8°.

At the post-mortem examination there was found much internal hydrocephalus, with opacity of the arachnoid at the base, but with no other evidence of acute meningitis. There was no sign of tuberculosis.

Case 30.—Edwin Thomas R—, aged 4½ months, admitted March 3rd, 1901, died May 31st, 1901.

On admission under Dr. Sansom the child had been ill for five weeks, and had had frequent convulsions. He had been fed at the breast; both mother and father were healthy. He was very irritable, and the head was markedly retracted.

On March 7th occasional retraction of the upper lids was noticed. On March 12th the head was still rigidly retracted, the fontanelle was found to be bulging, and there was occasional retraction of the upper lids. There was no rigidity of the limbs, and Kernig's sign was not present. There had been no convulsions since admission, and the child had taken his food fairly well, though there was occasional vomiting. The weight had decreased from 11 lbs. 6 oz. to 10 lbs. 10 oz. The fundi appeared normal.

The child continued in much the same condition, occasionally appearing to improve slightly and at other times appearing worse. It appeared that the child was blind, but no changes in the fundi were noted at any time. Kernig's sign was never present, but occasionally there seemed to be some slight rigidity of one arm.

Vomiting was infrequent after the first fortnight until a week before death, when it recommenced with some diarrhœa. The retraction of the head persisted throughout, and the occasional drawing back of the upper lids also continued. No strabismus was noted at any time. The temperature was normal or subnormal throughout, except on three occasions, when for a few hours it rose to 100° or 101°. Just before death the child weighed 9 lbs. 4 oz. Lumbar puncture was performed on March 12th, and the diplococcus intracellularis found present with another bacillus, resembling the bacillus influenzæ. The total duration of the illness was eighteen weeks. At the post-mortem examination all the organs except the brain were found to be healthy. The ventricles were enormously distended with fluid, and there was much fluid in the spinal theca. There was some milkiness of the membranes at the base, but no lymph was found anywhere.

Case 31.—Harry K—, aged 1 year, admitted February 28th, discharged July 12th, 1901. The child had been well until 10.30 p.m. on the day before admission under Dr. Gilbart Smith, when he became feverish and vomited. He had been breast-fed until admission, but occasionally had had a little cow's milk. There were signs of broncho-pneumonia with evidence of consolidation at the right apex. The temperature was 104·6°.

On March 7th a squint was noticed, and it was thought that there was some retraction of the head; the hands twitched occasionally. The body generally was somewhat rigid. The knee-jerks were not obtained. The signs of broncho-pneumonia had disappeared. On March 11th he vomited once; on the 14th there was marked retraction

of the head, and Kernig's sign was present; the knee-jerks were obtained. On March 15th lumbar puncture was performed, and the fluid was reported to contain the diplococcus intracellularis meningitidis. On the following day the notes say that the fontanelle was depressed; there was double internal strabismus; the pupils were equal and of medium size, and reacted to light. There was no rigidity of hands or arms or of neck; there seemed to be no definite evidence of Kernig's sign, though there was some resistance to complete extension of the knee-joints when the hips were flexed. There was no retraction of the upper lids. The child seemed to be getting much thinner, though he took his food well. The fundi showed no apparent pathological change.

On March 22nd the fontanelle was depressed; there was right internal strabismus, occasional retraction of the upper lids, and marked retraction of the head. There was some rigidity of the shoulders, and Kernig's sign was present. Since the 20th vomiting had been frequent, and he had taken his food badly. The fundi appeared normal. On March 27th the retraction of the head was still very marked, and the wasting was pronounced. There was no rigidity of the limbs, and Kernig's sign was not present. The pupils were equal, and there was right internal strabismus; the jaw was frequently moved as if in mastication. The vomiting continued, but for the last two days he had taken his food better.

On April 3rd the head was markedly retracted, and the limbs were kept flexed. The wasting continued, but since the 30th vomiting had been infrequent. The movements of the lower jaw were very marked; the squint persisted. The child lay half asleep, crying very little. The eyes were sometimes closed and sometimes open; occasionally the upper lids were retracted, so that the sclerotics showed above the corneæ. The fundi appeared normal.

On April 16th the emaciation was extreme, and the retraction of the head very marked; the lower jaw was constantly moving as if in mastication. The eyes were

kept open and the eyeballs were depressed, and retraction of the upper lids was very frequent. There was some rigidity of the legs, but Kernig's sign was not definitely present.

On April 20th the child seemed in much the same condition. There had been no vomiting since April 9th.

On May 1st the child seemed much better; he was very fretful if disturbed, and moved his limbs freely and spontaneously.

On May 3rd it was noted that the child looked fatter, and had gained 2 lbs. in weight in the past week. He seemed more fretful. There was depression of the eyeballs, with occasional retraction of the upper lids. The squint was still present, and also the frequent movements of the lower jaw. The girth of the head was 19 inches, an increase of half an inch since the last measurement on April 18th.

On May 9th and subsequently the girth of the head was 19¼ inches. From this time the child seemed steadily to gain ground so far as his general health was concerned, but he gradually got into a condition in which he lay upon his back all day kicking and throwing his arms about, and laughing in a manner suggesting a condition of idiocy. The depression of the eyeballs continued, and there developed a kind of coarse nystagmus or intermittent squint. The optic discs appeared whiter than normal; it was not thought, however, that the child was blind.

The temperature, which was 104·6° on admission, when there was broncho-pneumonia, fell two days later to 97·2°; after this it was irregular, varying between 97° and 103° for ten days, and then irregular with slighter variations for four more weeks, after which it was normal. The vomiting began on March 11th, and continued to be severe until April 29th, after which it was much less frequent and soon ceased.

On April 15th, when the emaciation was most extreme, the child weighed 13 lbs.; on discharge his weight was 1 st. 7 lbs.

Seen on December 3rd, 1901, the child was well nourished and happy; could walk well, though he carried his head tilted a little to the left. He could see and hear well. He could not be got to speak, however. Girth of head, 19½ inches. The child did not appear to be an idiot, or much below the normal mental development for his age.

Case 32.—William A—, aged 3 years, admitted September 17th, died September 28th, 1901. This child was admitted under the care of Dr. Percy Kidd, with the history that he had been vomiting frequently for the past fortnight. A squint had been noticed for one day; no retraction of the head or twitching of the limbs had been noticed.

On admission there was no rigidity of the neck, and Kernig's sign was not present; the child resented disturbance. There was some dissociated movements of the eyeballs, producing an occasional squint. The pupils were small, and the left was slightly larger than the right.

On September 20th there was some rigidity of the neck. On September 22nd lumbar puncture was performed, and from the fluid thus obtained Dr. Hunter isolated the diplococcus intracellularis.

For the first week in hospital the temperature was normal; it then rose, and remained between 99° and 101° until death on September 28th. There was no vomiting in the hospital. The duration of the illness was twenty-seven days.

At the post-mortem examination there was found some broncho-pneumonia at the base of the right lung, and a caseating bronchial gland. The other viscera appeared healthy. There was much purulent lymph at the base of the brain and on the under surface of the cerebellum; the convolutions were matted together. Tubercles were found along the vessels. The ventricles were distended with fluid, and in them was found some floating lymph. The vertex appeared normal save for the flattening of the convolutions.

Case 33.—William W—, aged 1 year 9 months, admitted March 13th, died March 17th, 1901. The child was first brought to the out-patient department on July 22nd, 1899, when three months old, and was then found to be suffering from congenital syphilis; there was a nasal discharge and the hot-cross-bun deformity of the skull. Up to that time he had been breast-fed, and was ordered to be given the bottle in addition. The child was under treatment until March, 1900, showing considerable improvement. Attendance was then discontinued until December 19th, 1900, when the child was again brought up with a history of irritability and retraction of the head; it seemed possible that the child had tuberculous meningitis. He was brought up again twice and improved, ceasing attendance on January 16th. On March 13th he was again brought up, having had a convulsion; there was then retraction of the head and strabismus; the temperature was 99°. He was admitted under the care of Dr. Percy Kidd. Shortly after admission he again became convulsed, both arms and the right leg being rigid and the left leg showing clonic movements. The eyes were turned to the left and there was no strabismus. Kernig's sign was present on both sides. No definite changes in the fundi were observed. Lumbar puncture was performed, and half a test-tubeful of clear fluid was removed. After this the convulsions ceased. On March 15th rigidity of the neck was noticed, with bulging of the fontanelle; there was no squint and no rigidity of the limbs; Kernig's sign was not present. There was no retraction of the upper lids.

On March 16th retraction of the head was marked, and there was difficulty in swallowing. Kernig's sign was present upon the right side only.

The temperature was 97° on admission, and rose to 101·8° on March 15th, falling to 99° on the 16th, thence rising rapidly to 108° just before death, on the morning of the 17th.

At the post-mortem examination there was found acute

miliary tuberculosis of all organs, with basal tuberculous meningitis.

Case 34.—Ethel G—, aged 3½ years, admitted April 15th, died April 19th, 1901. This child was first brought to the out-patient department on April 10th, with the complaint that she had been vomiting all food for the past five days. The bowels had not acted for two days, and there was headache. The temperature was 99·4°. The abdomen was a little retracted, but there were no definite physical signs of disease. The knee-jerks were obtained. Three days later the child was seen again. The temperature was 101°, the vomiting and headache continued, and the bowels had not acted. There was no retraction of the head, and Kernig's sign was not present; the knee-jerks were not obtained. On April 15th she was brought up again, and it was found that a squint had developed; the knee-jerks were not obtained, and there was some rigidity of the neck. The headache had persisted, and she had vomited once each day. She was admitted to the hospital under Dr. Percy Kidd. In the ward the temperature was 101·2°, and it was noted that there was an intermittent squint. Kernig's sign was not definite. On the following morning it was noted that the child lay usually with her eyes open, but that she could close them. The left pupil was larger than the right; both reacted to light; there were dissociated movements of the eyeballs, but no retraction of the upper lids. The neck was rigid, but there was no retraction of the head; the limbs were kept in the attitude of flexion, and she resisted attempts to straighten them, but Kernig's sign was not definitely present. Passive movements of the limbs seemed to cause pain. The fundi appeared normal; she was not blind, and there was no photophobia. On April 18th the child was much worse, being quite unconscious. Kernig's sign was definitely present in both legs. There were continued irregular movements of the arms and legs without any tendency to repetition, and more marked on the left side.

The neck was rigid, but there was no retraction of the head. There was no retraction of the upper lids. On April 19th it was noted that there were frequent dissociated movements of the eyeballs, with a kind of coarse nystagmus in the left eye, apparently produced by frequent contractions of the left internal rectus. The child seemed to be blind, and the fear reflex was absent. The right pupil was larger than the left, and neither reacted to light. There was no retraction of the lids; Kernig's sign was not present; the neck was rigid, but the head was not retracted. The knee-jerks were not obtained, possibly owing to the rigidity of the legs. There was considerable resistance to passive movement in all the limbs. The arms both carried out large, slow, irregular movements, without any tendency to a repetitive cycle. These movements were most marked in the left arm; the left leg also performed slight, irregular, slow, large movements. The note says that the movements were choreic in type, but were apparently modified by the rigidity of the limbs. The distribution was mainly hemiplegic. Lumbar puncture was performed on April 16th, and two test-tubes filled with clear fluid; there was apparently no amelioration of the symptoms. The temperature on admission was 101·2°, and rose to 102·9° just before death on the evening of April 19th. There was no vomiting in the hospital.

At the post-mortem examination there was found miliary tuberculosis of the lungs and spleen, with a caseous bronchial gland. There was much lymph at the base of the brain, and many tubercles were seen in the Sylvian fissures. There was an excess of fluid in the spinal theca, but no lymph; there was a slight excess of fluid in the ventricles.

Case 35.—Charles F—, aged 6 months, admitted April 16th, died April 21st, 1901. The child had been taken ill on April 8th with vomiting and restlessness; on the day of admission under Dr. Warner he had three

fits, which, according to his mother's account, were stronger on the right side. The child was breast-fed. The face was flushed, the head markedly retracted, and there were general twitchings of all the muscles of the body. The knee-jerks were present and equal. Kernig's sign was not obtained. The fontanelle bulged, but, on lumbar puncture, no fluid escaped. There were signs of general bronchitis.

On April 17th there was marked opisthotonos, the eyes were kept open, and there was occasional retraction of both upper lids, showing the sclerotics above the corneæ. There were dissociated movements of the eyeballs, but there was no persisting squint. The pupils were widely dilated and fixed. There was no photophobia, and the fundi appeared normal. The fontanelle bulged, and no pulsation was felt. There was no rigidity of the limbs, and Kernig's sign was not present. The knee-jerks were readily obtained. The breathing was periodic; at the end of each apnœic interval there was a spasmodic extension movement of all the extremities. There was no cyanosis.

On April 18th nasal feeding had to be commenced. On the 20th there were frequent convulsive seizures, the head and eyes being turned to the right, with movements of the extremities, which were more marked upon the right side. In the intervals there were constant clonic movements of the eyes to the right, producing a kind of coarse nystagmus. There was rigidity of the neck, but no retraction of the head; the arms were rigid, especially the right. Kernig's sign was obtained. The temperature on admission was 99·2°, and remained between 100·2° and normal until just before death, when it rose to 102·4°. There was frequent vomiting while the child was in the hospital.

Lumbar puncture was repeated on April 18th, and in the fluid obtained Dr. Hunter reported the presence of *Diplococcus intracellularis meningitidis* with other bacilli.

At the post-mortem examination a caseous bronchial

gland was found, and there was miliary tuberculosis of the lungs and spleen. There was much lymph at the base of the brain, and many tubercles were found both in the Sylvian fissures and down the spinal cord. The ventricles were distended with fluid.

Case 36.—John B—, aged 3, admitted April 20th, died April 28th, 1901. On admission under Dr. Gilbart Smith the child had been ill for two weeks with a bad cough, feverish, and loss of appetite; there had been diarrhœa, but no vomiting. There was no history of tuberculosis in the family. The temperature was 101°.

On April 22nd the child appeared drowsy, sleeping with his eyes shut and taking notice of nothing save actual disturbance, which he resented. Passive movement of the limbs was resented and seemed to cause pain; Kernig's sign was not present. There was no retraction of the head and no squint; the pupils were equal and reacted to light; the fundi were normal. Vomiting was frequent, and he took his food badly. There were no signs of thoracic or pulmonary disease.

On April 23rd the neck was rigid, but the head was not retracted; the pupils reacted sluggishly to light, and the left was larger than the right. The legs were stiff, but Kernig's sign was not present; the knee-jerks were not obtained.

On April 26th the child was in a comatose condition, in no wise resenting interference; there was a blotchy, purplish flush upon the face. The left eye was closed and the right open; the left pupil was dilated and fixed; the right was smaller and reacted to light. There was marked rigidity of the right arm and hand, with all the joints fully flexed. The joints of the left arm were also flexed, but were not so rigid. The left hip and knee were flexed, and the ankle extended with considerable rigidity; Kernig's sign was obtained upon the left side. Nasal feeding was necessary.

Lumbar puncture was performed on April 27th, and

half an ounce of fluid was obtained, in which the diplococcus intracellularis meningitidis was found by Dr. Hunter.

The temperature was 101° on admission, and remained at about 101° until just before death, when it rose to 104·2°.

At the post-mortem examination there were found caseous bronchial glands and miliary tubercles in lungs and spleen. At the base of the brain there was much greenish lymph, with numerous miliary tubercles. There were no tubercles on the cord. The ventricles and spinal theca were distended with clear fluid, and there was much clear fluid in the basal arachnoid cistern.

Case 37.—Lily G—, aged 10 months, admitted April 3rd, died April 13th, 1901. The child, who was breast-fed, seemed healthy until March 24th, when she had an attack of vomiting with some diarrhœa, which lasted for three days. On March 30th a squint was noticed, and her mother said that the eyes "rolled about." She became drowsy and screamed at times. She was brought to the hospital on April 3rd, and admitted under Dr. Gilbart Smith. The neck was then rigid, but there was no retraction of the head; the fontanelle was not bulging. There was a squint. The pupils were equal and reacted to light; the fundi appeared normal. Kernig's sign was not present; the knee-jerks were equal and increased.

On April 9th nasal feeding had to be commenced, but the child vomited after each feed. There was no retraction of the head, and the fontanelle was slightly depressed; there was some strabismus. The limbs were not rigid, and Kernig's sign was not present. There was no retraction of the upper lids.

On April 10th lumbar puncture was performed, and 3 oz. of clear fluid removed, and was reported to contain the meningococcus. After the lumbar puncture there seemed to be definite improvement, which, however, only lasted for a short time, and in about forty-eight hours she seemed to be worse again.

On April 12th the left pupil was larger than the right,

and neither reacted to light. The child lay taking notice of nothing, and not resisting disturbance in any way.

The temperature on admission was 99·8°; it continued to be irregular between 97° and 99·4° until just before death, when it fell to 96·6°. Vomiting continued throughout, and towards the end there was some diarrhœa.

At the post-mortem examination caseating bronchial glands, tuberculous broncho-pneumonia, and miliary tuberculosis of the spleen were found. The membranes at the base of the brain were thickened and studded with miliary tubercles. There was no excess of fluid in the lateral ventricles, the walls of which were slightly roughened with a thin layer of lymph.

Case 38.—Ellen H—, aged 5, admitted June 17th, died June 27th, 1901. The child was brought to the out-patient department on June 13th, with the history that she had felt "tired" for the last fortnight, and that she had felt worse for five days, and had got a headache. She had vomited once on June 12th, and there had been constipation for three days. The temperature was 100°. There was not then found to be evidence of organic disease, and she was sent home with an aperient mixture, to come again in four days. When she returned the temperature was 101·4°, and she seemed worse and was consequently admitted into the hospital under Dr. Gilbart Smith.

On admission, beyond the fever and the wasting there were no physical signs of disease. The fundi were normal. On the following day it was noted that the child was very irritable and complained of headache; the neck was rigid, but the head was not retracted; Kernig's sign was obtained on the left side only. Lumbar puncture was performed, and 3½ oz. of clear fluid removed, which proved to be sterile. The withdrawal of the fluid seemed to give immediate relief; the irritability ceased, and the child went to sleep while the fluid was still draining.

On June 19th the child seemed drowsy; the upper lids drooped, and there was a definite squint; the tongue was

foul and there were sordes on the lips. Taches bleuâtres were found scattered over the trunk. Kernig's sign was definitely present on both sides.

On June 21st the child was still drowsy; she was irritable when disturbed, and cried out at times, "My poor head." The neck was rigid; the pupils were dilated and their reaction to light doubtful. There was a definite squint; Kernig's sign was present on both sides; the knee-jerks were not obtained.

On June 25th it was noted that the child seemed worse; she rolled her head from side to side, and there were irrelative movements of the arms. The eyes were kept open, and occasionally the upper lids were retracted. Dissociated movements of the eyeballs were noticed, and the child was apparently blind. The neck was still rigid, but there was no retraction of the head. The fundi appeared normal. The temperature remained between 100° and 101° for a week, and then sank to normal, continuing so (with one slight rise) until death on June 26th.

The post-mortem examination showed acute miliary tuberculosis with tuberculous meningitis.

APPENDIX II.

This case is recorded separately as an example of chronic hydrocephalus sequent upon acute meningitis. It was not included with the other cases because the presence of pulmonary tuberculosis might conceivably invalidate any deductions drawn from a list in which it was included.

A case of simple meningitis with pulmonary tuberculosis.—Henry W. P—, aged 8 months, admitted February 6th, 1902, died March 19th, 1902. This child, one of twins, was admitted under the care of Dr. Sansom with the history that he had been wasting for four months, and that for three months the head had been increasing in size. For the first month he had been breast-fed. The other twin died when four months old. Two relations on the mother's side had suffered from hydrocephalus; one

died at the age of ten; the other is still living, and is aged twenty. The child, except for the size of the head, seemed fairly healthy and happy; both anterior and posterior fontanelles were widely patent and bulging. The measurements of the head were: occipito-glabellar, 12 inches; transverse (ear to ear), 12·5 inches; circumference, 18 inches. The heart and lungs appeared normal. For the first fortnight the temperature remained normal. On admission lumbar puncture was performed, and from it Dr. Ainley Walker isolated a diplococcus which he considered to be identical with that described by Weichselbaum, both in morphological and cultural reactions. A week later the child commenced to vomit at intervals, and a fortnight after admission the temperature rose and continued to be irregular (99°—106°) until death four weeks later. On February 22nd nystagmus developed, which seemed to be rotatory in the left eye and lateral in the right eye. On March 7th a copious discharge of clear cerebro-spinal fluid began from each ear, and continued until the end. There was no rigidity of the neck throughout, and Kernig's sign was never obtained. Death took place from gradual exhaustion. The total duration of the illness was at least 4½ months, dating from the first noticeable enlargement of the head.

At the post-mortem examination the body was found to be extremely wasted; the head appeared large in comparison with the body; the anterior fontanelle was widely patent, and the sutures separated, but not widely. At the root of the right lung there was a small caseous focus, proved microscopically to be of tuberculous origin, and there were a few scattered grey tubercles near the base of the left lung. There was a caseous bronchial gland. No evidence of tuberculosis was found in any other part of the body. There was found to be a large excess of clear fluid in the cerebral ventricles, at the base of the brain, and in the spinal theca. There was some thickening of the arachnoid in the interpeduncular space, and about the superior verm of the cerebellum. There was no evidence

of thickening or matting of the membranes in the region of the roof of the fourth ventricle. No lymph and no tubercle was found either upon the brain or cord. The lateral ventricles were very greatly dilated, so that they occupied the greater part of each hemisphere; the third and fourth ventricles were also much dilated, but the iter did not seem much larger than is normal. The central canal of the cord was not dilated. The veins on the walls of the lateral ventricles were distended; the choroid plexus was not distended or thrombosed. There was no evidence of thrombosis in any cerebral sinus. Both petrous bones appeared healthy. The brain substance was soft, but presented no other abnormality.